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Archives of Internal Medicine

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BODY WEIGHT IN TWO HUNDRED AND TWENTY-NINE ADULTS

WHICH STANDARD IS THE BEST?

H. GRAY, M.D., AND J. F. MAYALL

BOSTON OXFORD, MASS.

PROBLEM

It is rare for clinicians to recognize that medicine, as a whole, profits more by normal standards than by scattered observations of interesting pathologic cases. This is Benedict's judgment,[1] and Galton's [2] appears to have been much the same: "Variations of weight are the surest guides to health. . . . Dangerous illnesses may be avoided, and even life preserved." Such illnesses may be outspoken, like malnutrition in children, thyrotoxicosis and tuberculosis; also diabetes and obesity. Or the disease may be insidious, in which case the weight abnormalities are apt to be called defects without obvious cause. Such defects have been alarmingly frequent in recent military examinations. And not only frequent, but surprisingly important. For instance, if we are conservative and consider not the incidence among draftees but only the actual rejections, we find that overweight ranks second among the prominent causes of rejection, as detailed in Table 1.

TABLE 1.—PHYSICAL CAUSES OF REJECTION

Causes of Rejection in Order of Frequency	By Seven Local Boards for the Army[3]	By the Navy and Marine Corps[3]	By the Total Draft for the Army[4]
	No. examined.....7,611	No. examined.....82,592	No. examined..2,510,591
	Percentage of Examinees Rejected	Percentage of Examinees Rejected	Percentage of Examinees Rejected
1	Eyes..... 6.0	Eyes..... 11.4	Heart..... 4.3
2	Teeth..... 4.8	Underweight..... 6.5	Underweight..... 2.6
3	Underweight..... 4.6		
15 etc.	Overweight..... 0.4	Overweight not given	Obesity..... 0.17

The literature furnishes an embarrassing variety of standards by which to judge whether the weight of a given person is within normal

1. Benedict, F. G.: Boston M. & S. J. **181**:109, 117, 118 (July 31), 1919.
2. Galton, F.: Life History Album, London, 1884, p. 2.
3. Fisher, I., and Fisk, E. L.: How to Live, Ed. 15, New York and London, 1919, p. 400, 258.
4. Love, A. G., and Davenport, C. B.: Defects Found in Drafted Men, Printed for Senate Committee on Military Affairs, Washington, 1919, p. 71.

limits. To enable the physician or statistician to choose between these methods is the main object of this paper. The plan has been to collect measurements and actual weights on a series of normal individuals, then to predict the weights for these men by means of various standards, and finally by comparing these predicted weights with the actual weights to compute the error made by each standard.

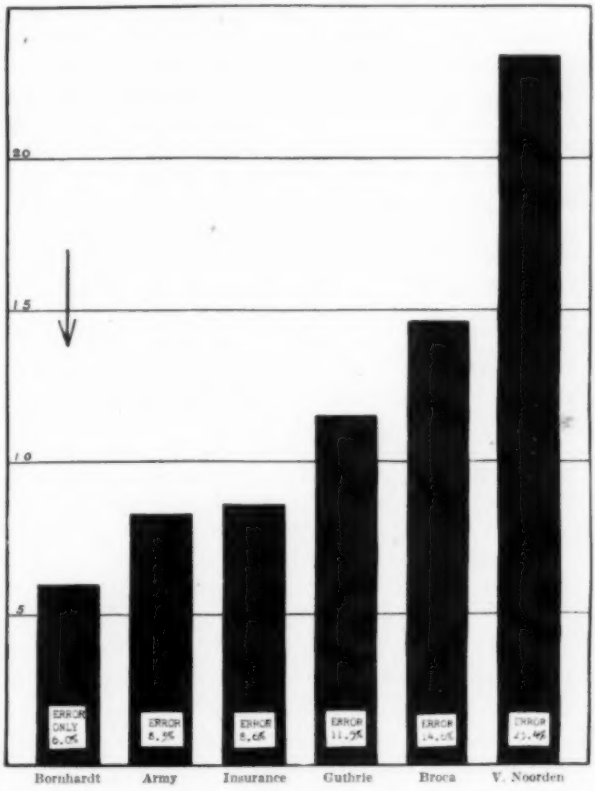
INDIVIDUALS OBSERVED

The first series was abruptly terminated by entry into service of one of the authors, and was, therefore, reported at the time.[5] It is summarized in Table 7, together with some new calculations based on the original data. But this earlier series comprised only twenty subjects, and so it was thought desirable to assemble a second group. This was done in the intervals between regular work in the U. S. Army Base Hospital 76, mostly in France, while the extensive computations have been carried out since leaving the service. This second series comprises 229 soldiers, all from the personnel of Base Hospital 76. They were all born in the United States, mostly in Maryland and Pennsylvania. Most of them were inducted at Fort Oglethorpe in June, 1918, and mobilized as this hospital unit at Camp Devens in July, 1918. At that time they were weighed naked, measured and examined physically by the medical officers of the unit, in the hope of excluding some on the ground of possible defects overlooked in the hastier examination at enlistment. Incidentally, the number of positive Wassermanns in 200 tests was only one. The unit sailed from New York August 31, spent about a week in England, and was on duty in Vichy, Allier, France, from the latter part of September until after the time of the second weighing in January, 1919. Some of the original members had meanwhile been separated from the unit, but all who had been weighed twice at the six months' interval are included in this report. Nobody was excluded by preconceived notions on our part as to the typical man. The series is, therefore, unselected, except that males of military age who happen not to fit the army standard are not represented. This fact constitutes a technical defect, as pointed out by Hoffman, but the advantage accrues to a method other than that championed in this paper. Accordingly, we have assumed these to be fair samples of American-born young men, and as such to be suitable controls by which to test the accuracy or error of different methods for predicting weight.

The gist of this test is that Bornhardt's standard is the most accurate, closely followed by the Army and insurance tables; while

5. Gray, H., and Gray, K. M.: Normal Weight, Boston M. & S. J. **177**:894 (Dec. 27) 1917.

Guthrie's, Broca's and von Noorden's give much less satisfactory results. This is shown graphically in the accompanying chart, and is detailed numerically in Table 9. The casual reader, therefore, may well omit the following minutiae as to method, and skip to the conclusions at the end of the paper.



Average error in predicting weight by different standards. Difference between predicted and actual weights, expressed as percentage of the actual weight.

DEFINITIONS

The actual weight is often represented by such terms as: net weight, weight stripped, weight without clothes. This is the criterion by which we are to choose between various standards in the literature.

The Weight of Clothes.—Nearly every standard except the medico-actuarial is expressed as net weight. In our previous observations [5]

men's clothing averaged eight pounds (3.6 kg.), and heels averaged one inch (2.5 cm.). By these values the medico-actuarial table, derived from life insurance records, has been adjusted for net weight in making the comparisons both in the earlier series and the present series. It would have been more accurate, however, for us to have subtracted not 8, but only 6 pounds (2.7 kg.) from the medico-actuarial table to find the predicted weight, because that table was based on weighings with clothes and shoes, but without coat and vest. If, now, we should correct by an increase of 2 pounds our weights predicted by that table, they would be mathematically more correct, but would make no better showing for that standard, since the predictions already run too high. In practice, when using any other than the insurance table, the average weight of house clothes and shoes for men at all ages, sizes, and seasons may be allowed for by Table 2. The figures opposite Quetelet's [6] name were obtained by applying his rule, that clothes average one-eighteenth of the total weight of the male body, to the average weight for the age group here considered, i. e., from 20 to 34 years inclusive (not the average weight for our own group, which is so small relatively and gives 7.8 pounds, i. e., 3.5 kg., but) as computed by us from figures given [9] for 130,301 men insured in the United States and Canada: 152.1 pounds (69.1 kg.) with clothes; then clothes come to 152.1/18 = 8.5 pounds (3.8 kg.).

TABLE 2.—Allowance for Men's Clothes and Shoes *

Writer	Weight of Clothes and Shoes	
	Kg.	Lbs.
Fisher and Fisk	3.1	6.9
Gray	3.6	8.0
Quetelet	3.8	8.5
Gaertner[7]	4.0	8.8
Harris and Benedict[8]	4.0	8.8

* Expressed as kilograms or pounds.

The *ideal weight* is that accompanied by the healthiest or longest life. Too few people are familiar with the insurance experience [3] that one should endeavor to keep his weight at the average for his height and for age 30, the period of full maturity.

6. Quetelet, L. A. J.: Sur l'homme, Paris, 1835, **2**:44, or in the first English translation, A Treatise on Man, Edinburgh, 1842, p. 64.

7. Gaertner, G.: Diätetische Entfettungskuren, Leipzig, 1913, pp. 154, 158, 160; or in English translation, Reducing Weight Comfortably, Philadelphia, 1914, pp. 278, 286, 291.

8. Harris, J. A., and Benedict, F. G.: A Biometric Study of Basal Metabolism in Man, Carnegie Institute, Washington, Publication No. 279, 1919, pp. v, 2, 3, 4, 7, 21, 26, 163, 164, 228.

9. Medico-Actuarial Mortality Investigation, Vol. I, Statistics of Height and Weight of Insured Persons, published by Assn. Life Ins. Directors and Actuarial Soc. of America, N. Y., 1912, pp. 13, 16, 18, 38, 67, 105, 109.

The *average weight*, or usual weight, is the familiar arithmetic mean weight, of subjects grouped by age, height, or other more complex methods. In the medico-actuarial investigation the average weight agreed closely with the central weight (also technically called the mode, the most frequent or the typical weight), thus showing the reliability of using the average.

The *correct weight* may signify either the ideal or the average weight, hence unfortunately it leads to ambiguity.

The *normal weight* also may be interpreted as either the ideal or the average weight. For children Holt [10] says that "the average is not to be confused with the normal. There are considerable variations on either side of the average which should be regarded as within the range of normal. The normal is a zone, not a line." Benedict,[1] too, believes that "for the growing child a gross error has been committed in . . . accepting the average . . . as normal." For adults the insurance committee makes a like statement: "Normal weight for any age and height may vary considerably from the average."

Normal Zone Weight.—This term might be used for the healthy range called "standard lives" by the insurance committee, "the norm of weight" by Gaertner, and more recently emphasized by Holt in the words cited above. The phrase is suitable to express any weight within acceptable limits, but yet not the ideal nor the exact average. The lower boundary of this zone, i. e., the minimal permissible figure beneath which the person is judged underweight, is given by Emerson [11] as 7 per cent., and by Holt as 10 per cent., both for children. For adults, figures which we have calculated from the 1916 printing of the Army standard give 9 per cent. (ranging from 6 to 13 per cent. according to height), and from the 1919 edition 13 per cent. (ranging from 8 per cent. acceptable below the standard weight for 60 inches, up to 19 per cent. below the standard weight for 74 inches); while the insurance companies regarded lives as standard even down to 15 per cent. underweight.

The *standard weight* may mean the ideal, or the average, or the zone.

A *weight standard* is any method, whether table or formula, for predicting weight, as in the title to this paper.

Pathologic Weight, Underweight, Overweight.—Whatever term be used, whatever standard be selected, whatever variation above and beneath the average be accepted as healthy, nearly every writer on obesity or malnutrition starts his reckoning from the predicted weight.

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10. Holt, L. E.: Am. J. Dis. Child. **16**:359 (Dec.) 1918.

11. Emerson, W. R. P.: Am. J. Dis. Child. **17**:251 (April) 1919; Boston M. & S. J. **181**:139 (July 31) 1919.

Predicted weight, calculated weight, estimated weight, expected weight, theoretical weight. This value may be derived from a variety of tables and formulae, of which six are analyzed. It usually corresponds to the average weight.

STANDARDS COMPARED

While the average weight is affected by various factors, e. g., age, sex, country and locality of birth, height, chest-girth, individual variation or sampling; the weight has in nearly every standard offered been considered in connection with one of the following four periods of life: Birth, preschool age, school age, or adult life. The borders of these four groups, except of course the first, vary somewhat according to different writers, but this is of no concern here. For each of the first three age-groups one table was chosen and reproduced in a previous paper;[5] while for the last or adult age group six standards were quoted: Two tables and four formulas. They were Bornhardt's, the Army's, the medico-actuarial, Guthrie's, Broca's and von Noorden's.

Bornhardt's formula was used in the shape given by Fröhlich,[12] Vierordt,[13] Gaertner,[7] Baer,[14] Barker [15] and Vedder,[16] namely: If H be the height without heels in centimeters, C the mean chest girth in centimeters as measured over the nipples, W the net weight in kilograms, then the expected weight for the adult of average constitution is: $W = H \times C/240$.

The Army Table [17] used in our earlier paper [5] has been adhered to in the present study, for the sake of uniformity. The editions of "the physical examination standards . . . are . . . all together seven in number," [18] from G. O. 66, A. G. O., April 18, 1910, to the latest and larger table authorized Nov. 8, 1918, and published in Washington, 1919. The changes have been slight.

The *Navy Table* [19] gives the same weights for the same heights, i. e., from 64 to 73 inches inclusive, as in the edition of the Army table used here.

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12. Fröhlich, H.: Allgem. medic. Central. Zeitg. **64**:8 (Jan. 2) 1895.
13. Vierordt, H.: Tabellen für Mediziner, Ed. 3, Jena, 1906, p. 97.
14. Baer, J.: Fettsucht (Obesity), in L. Mohr and R. Staehelin's Handbuch der inneren Medicin, Berlin **4**:650, 1912.
15. Barker, L. F.: Clinical Diagnosis of Internal Dis., New York and London **3**:794, 1916; or Monographic Medicine **4**:794, 1916.
16. Vedder, E. B.: Sanitation for Medical Officers, Medical War Manual No. 1, Ed. 2, Philadelphia, 1918, p. 74.
17. McCain, H. P., Adjutant General, War Department, U. S. A. Recruiting Circular No. 2, Washington, Nov. 1, 1916, i. e., G. O. 66 W. D.
18. Hoffmann, F. L.: Army Anthropometry, Prudential Press, Newark, N. J. **34**:46, 1918.
19. Bureau of Navigation, U. S. Navy, Circular Relating to the Enlistment of Men, July 20, 1912, also Circular N. M. S. 126,480, October, 1916; also Manual for the Medical Department, U. S. Navy, 1917, par. 2082.

The *Medico-Actuarial Table* is not reproduced, being very long and available elsewhere.[5, 9, 20]

Broca's Formula, so-called,[15] has also been credited to Quetelet [21] by some,[22] to Allaire [23] and to Robert [24] by others,[7, 25] and cited without credit by still others.[26] We have not succeeded in finding the original, despite search of the supposed sources. The formula is uniformly expressed as: W (in kg.) = H (in cm.) minus 100. Bouchard believes the rule to be approximately true for heights between 168 and 175 cm.; but we find, from applying the rule to the 116 men of our series who fall within this restricted range, that the prediction error, without regard to sign, averages 14 per cent. This is not materially better than the average for our whole series which will be seen in Table 8.

Guthrie's Formula, worked out on patients in Professor Barker's clinic,[15] is: Wt. in pounds = 110 + (5.5 × [ht. in inches minus 60]).

Von Noorden's Formula is given by Barker as: Wt in kg. = ht in cm. × 430 to 480. If from this statement the constant factor for average weight be inferred to correspond to the mean between these two values we get: Wt in kg. = ht in cm. × 455. This interpretation has been applied both in the earlier paper and in this. The results cause regret that we have not been able to see Von Noorden's original statement of his rule.

OBSERVATIONS

The method of recording measurements was as follows: Weight to the nearest half-kilogram (or pound), height and chest-girth to the nearest centimeter, and age to the nearest year.[8, 9, 17, 19, 27] Harris and Benedict [8] point out that "A measurement of stature to the nearest centimeter is about the limit of accuracy. To retain tenths of kilograms is . . . a degree of refinement . . . Finally, when individuals are recorded to the nearest year of age we may remember that they are on the average a quarter of a year older or younger than the age to which they are assigned." Our technic also conformed in many respects to the recommendations of the Committee on Anthropology of the National Research Council.[18] For height a tape 150 by 1.5 cm. (59 x 5/8 in.) was tacked to the wall so that the lower end was 100 cm.

20. Joslin, E. P.: Diabetic Manual, Ed. 2, Philadelphia, 1919, p. 115.
21. Quetelet, L. A. J.: Anthropométrie, Bruxelles, 1871, p. 178, and elsewhere.
22. Sahli, H.: Diagnostic Methods, trans. from German Ed. 5, by N. B. Potter, Ed. 2, Philadelphia, 1911, p. 28. Heckel, F.: Grandes et Petites Obesités, Paris, 1911, p. 105.
23. Allaire: Receuil de mémoires de médecine milit., III e S. **10**:161, 1863.
24. Robert: Receuil de mémoires de médecine milit, III e S. **10**:171, 1863.
25. Fröhlich, H.: Körpergewicht, in Real-Encyclopädie der ges. Heilk., Ed. A. Eulenburg, Vienna, 1887, **11**:199.
26. Bouchard, C.: Traité de Pathologie Générale, Paris, **3**:183, 1900.
27. Treasury Dept., Bureau of War Risk Insurance Form 2A.

above the floor. The subject stood in bare feet, touching the wall with heels, hips, shoulders and head; and a block with square end was pressed down firmly on the scalp. For chest-girth a similar tape was applied on a level with the nipples and read at full inspiration and full expiration; midway between these two readings was taken as the mean chest-girth. The above technic is somewhat more precise than that considered adequate by the Medico-Actuarial Committee:[9] height to the nearest inch. Incidentally, if inches be preferred to centimeters, the inch may well be divided, not into eighths (binary fractions) as in ordinary rules, but into tenths. Similarly, fractions of pounds are better noted as tenths than as sixteenths (ounces). These decimal fractions occupy less space in the chart, are more quickly written down, and are better adapted for averaging and other statistical manipulation, as pointed out by Roberts[28] long ago.

The basal data obtained by our measurements are reported in Table 3, with the subjects arranged according to their actual weights, the lightest first.

METHODS OF STATISTICAL ANALYSIS OR CALCULATIONS

The importance of giving attention to methods considered fussy by some, has been urged by Harris and Benedict in these words: "Data must be . . . interpreted as the statistical constants . . . indicate without any regard to opinions heretofore expressed. In the past, data have been treated almost exclusively by the simple method of averages and graphic representation. . . . By means of the biometric formulas . . . during the past two decades instances of the demonstration of law and order in processes hitherto apparently chaotic have been rapidly multiplying. The reader who is interested in end-results rather than in methods should pass over these features, just as the general biologist must pass over the details of method. . . . in a paper by an organic chemist, realizing that they are essential to the technical development of the subject."

Pearl[29] more recently wrote a paper "to illustrate by a concrete example, not only the importance, but indeed the absolute necessity of mathematical tests of the validity of results and conclusions, if medicine is ever to measure up the standards of scientific logic and accuracy which prevail in some, at least, of the other branches of biologic and physical science. Many, indeed most, of the problems of practical

28. Roberts, C.: Manual of Anthropometry, London, 1878, p. 26. For example, see Reference 5 in which Table 2 reproduces the American Medical Association's table for children (first published by Crum) with the common fractions translated by us into decimals.

29. Pearl, R.: A Statistical Discussion of the Relative Efficacy of Different Methods of Treating Pneumonia, Arch. Int. Med. **24**:398 (Oct.) 1919.

TABLE 3.—OBSERVED DATA

Number	Name	Age at Nearest Birthday, in Years	Height Without Shoes		Chest Girth at Nipple, Mean, in Cm.	Actual Weight, Stripped		
			In.	Cm.		July, 1918, in Lbs.	Jan., 1919, in Lbs.	Per Cent. Gain (+) or Loss (−)
1	2	3	4	5	6	7	8	9
1	P. A. N.	25	60	152	88	105	120	+14
2	J. N. N.	22	66	168	83	107	105	— 2
3	J. M. W.	31	65	165	84	109	120	+10
4	P. J. S.	23	65	165	84	109	136	+25
5	M. L.	24	62	157	80	110	117	+ 6
6	S. S.	25	66	168	82	110	117	+ 6
7	M. K.	22	62	157	83	112	112	0
8	J. K. T.	22	61	155	82	113	126	+12
9	G. W.	30	65	165	89	114	126	+11
10	T. W. S.	23	64	163	88	116	122	+ 5
11	P. C. F.	25	63	160	84	116	124	+ 7
12	A. J. D.	22	62	157	86	116	125	+ 8
13	W. L. D.	28	65	165	86	116	139	+20
14	F. W.	22	64	163	90	117	129	+10
15	J. H. M.	28	63	160	84	118	120	+ 2
16	W. C. W.	28	63	160	86	118	126	+ 7
17	S. A.	27	62	157	85	119	125	+ 5
18	J. S.	26	65	165	86	120	120	0
19	M. J.	24	65	165	90	120	121	+ 1
20	S. Y.	28	64	163	86	121	129	+ 7
21	H. D.	25	68	173	90	121	129	+ 7
22	W. S.	26	58	147	80	122	118	— 3
23	J. P. V.	26	65	165	89	122	122	0
24	J. P. D.	27	63	160	90	122	122	0
25	L. W.	27	64	163	90	122	122	0
26	G. D. W.	33	66	168	80	122	126	+ 3
27	J. J. M.	23	66	168	88	122	133	+ 9
28	A. L. M.	28	71	180	85	122	135	+11
29	J. C. T.	27	66	168	90	123	127	+ 3
30	W. W. W.	27	69	175	90	123	130	+ 6
31	C. O. N.	27	68	173	84	123	137	+11
32	W. J. W.	23	62	157	91	123	140	+14
33	J. F. M.	33	69	175	85	24	129	+ 4
34	M. E. J.	19	69	175	80	124	133	+ 7
35	P. W. E.	26	67	170	81	124	130	+12
36	J. A. F.	24	67	170	88	124	146	+18
37	J. O.	21	65	165	89	125	132	+ 6
38	J. A. W.	25	66	168	93	125	132	+ 6
39	L. S.	22	65	165	90	125	136	+ 9
40	L. C. B.	26	65	165	85	125	144	+15
41	D. E. F.	24	62	157	89	126	128	+ 2
42	L. H. W.	27	66	168	90	126	131	+ 4
43	R. T. F.	23	69	175	89	126	134	+ 6
44	P. S. H.	24	69	175	83	126	137	+ 9
45	A. F. S.	26	65	165	89	128	132	+ 3
46	J. W.	29	68	173	90	128	134	+ 5
47	J. B. R.	23	67	170	80	128	136	+ 6
48	L. L. H.	25	68	173	84	128	141	+10
49	J. K.	23	64	163	86	128	141	+10
50	F. A. C.	30	69	175	90	128	141	+10
51	A. E. B.	31	69	175	86	128	143	+12
52	L. G. M.	23	65	165	88	128	146	+14
53	P. P. L.	32	69	175	85	129	142	+10
54	J. M. D.	23	69	175	85	129	145	+12
55	B. L. F.	23	64	163	89	129	150	+16
56	A. B. R.	28	67	170	91	130	130	0
57	P. J. S.	23	63	160	85	130	131	+ 1
58	R. M.	23	69	175	88	130	135	+ 4
59	O. L. O.	22	63	160	88	130	138	+ 6
60	E. S.	28	66	168	88	130	139	+ 7
61	F. J. F.	30	65	165	88	130	140	+ 8
62	P. R. C.	25	68	173	93	130	150	+15
63	A. B. B.	34	70	178	88	130	154	+18
64	F. S.	24	66	168	86	131	134	+ 3
65	C. M. A.	24	63	160	88	131	135	+ 3
66	V. P. B.	31	71	180	88	131	140	+ 7
67	J. E. J.	26	63	157	90	131	142	+ 8
68	G. F. M.	26	70	178	88	131	144	+10
69	W. S.	22	66	165	90	131	146	+11
70	O. C. M.	25	66	168	84	132	142	+ 8
71	J. P.	29	66	168	86	132	151	+14
72	H. R. S.	21	67	170	87	132	152	+15
73	M. W.	27	68	173	91	133	133	0
74	V. S. D.	29	66	168	86	133	134	+ 1
75	W. D. J.	23	69	175	89	133	135	+ 2
76	F. W. F.	23	67	170	91	133	140	+ 5
77	W. S.	28	65	165	93	133	142	+ 7

TABLE 3.—OBSERVED DATA—*Continued*

Number	Name	Age at Nearest Birthday, in Years	Height Without Shoes		Chest Girth at Nipple, Mean, in Cm.	Actual Weight, Stripped		
			In.	Cm.		July, 1918, in Lbs.	Jan., 1919. in Lbs.	Per Cent. Gain (+) or Loss (—)
1	2	3	4	5	6	7	8	9
78	W. C. B.	23	68	173	93	133	142	+ 7
79	L. P. A.	26	63	160	91	133	145	+ 9
80	A. B. C.	23	71	180	82	133	150	+13
81	C. L. V.	28	68	173	85	134	144	+ 7
82	M. M.	27	67	170	87	134	145	+ 8
83	L. G.	23	66	168	85	134	148	+10
84	J. L. B.	25	71	180	85	134	148	+10
85	F. M. S.	23	65	165	89	134	152	+13
86	H. W. D.	31	67	170	89	135	133	— 2
87	F. K.	22	68	173	89	135	135	0
88	F. J. D.	27	67	170	89	135	137	+ 2
89	J. M. T.	22	67	170	82	135	139	+ 3
90	M. R. D.	30	64	163	91	135	139	+ 3
91	L. S.	27	66	168	86	135	141	+ 4
92	E. J. C.	32	67	170	91	135	141	+ 4
93	W. J. M.	23	70	178	90	135	148	+10
94	H. A. U.	24	70	178	91	135	148	+10
95	F. N. B.	27	68	173	88	135	150	+11
96	B. J. S.	27	64	163	93	135	150	+11
97	E. H. W.	22	67	170	87	135	152	+13
98	R. O. G.	25	71	180	85	135	153	+13
99	J. S.	30	65	165	88	136	133	— 2
100	J. P. M.	29	67	170	93	136	144	+ 6
101	W. E. W.	28	65	165	88	136	145	+ 7
102	J. A. B.	33	67	170	90	136	149	+10
103	G. S. S.	25	68	173	89	136	152	+12
104	O. E. T.	23	69	175	90	136	152	+12
105	F. J. S.	25	69	175	86	136	153	+13
106	C. J. A.	27	68	173	90	136	156	+15
107	G. D.	28	66	168	89	136	157	+15
108	A. Z.	26	65	165	93	137	148	+ 8
109	S. J. H.	24	69	175	97	137	152	+11
110	J. B.	25	69	175	86	137	161	+18
111	F. J. F.	25	68	173	90	138	121	—12
112	S. J. H.	25	67	170	94	138	139	+ 1
113	J. R. K.	23	67	170	91	138	146	+ 6
114	J. W. B.	23	71	180	85	138	150	+ 9
115	E. S.	25	66	168	94	138	152	+10
116	W. J.	27	66	168	94	138	152	+10
117	C. F. S.	23	68	173	87	138	157	+14
118	E. R. W.	25	68	173	88	139	140	+ 1
119	J. A. A.	25	67	170	89	139	144	+ 4
120	P. H. O.	25	68	173	88	139	146	+ 5
121	J. M. C.	21	72	183	94	139	152	+ 9
122	H. H.	28	68	173	83	140	132	— 6
123	D. K. T.	18	68	173	86	140	137	— 2
124	R. E. M.	24	70	178	91	140	140	0
125	R. E. H.	21	69	175	86	140	145	+ 4
126	A. C. W.	28	70	178	87	140	142	+ 9
127	W. N. S.	26	70	178	88	140	154	+10
128	A. F. S.	28	67	170	92	140	174	+24
129	Z. E. C.	31	72	183	89	141	152	+ 8
130	G. W. K.	24	71	180	91	141	158	+12
131	J. N	24	68	173	90	141	168	+19
132	J. A	28	65	165	91	142	135	— 5
133	C. T.	28	71	180	91	142	142	0
134	P. K.	26	69	175	91	142	144	+ 2
135	H. E. G.	24	63	160	94	142	144	+ 2
136	L. A. M.	21	72	183	90	142	153	+ 8
137	F. R. P.	28	72	183	91	142	153	+ 8
138	M. J. R.	28	68	173	86	143	138	— 3
139	J. S.	27	67	170	90	143	142	— 1
140	W. C. S.	30	66	168	83	143	150	+ 5
141	J. J. L.	22	70	178	91	143	151	+ 6
142	A. W. H.	28	72	183	91	143	152	+ 6
143	P. F.	28	75	191	94	143	155	+ 8
144	L. A .D.	28	71	180	91	143	157	+10
145	G. H. S.	23	64	163	90	144	143	— 1
146	J. W. T.	31	69	175	93	144	144	0
147	K. R.	23	66	168	98	144	156	+ 8
148	W. H. M.	23	71	180	90	144	164	+14
149	J. C. M.	25	71	180	86	144	165	+15
150	V. V.	26	63	160	99	145	146	+ 1
151	L. D. O.	25	70	178	91	145	147	+ 1
152	C. H. F.	23	69	175	90	145	159	+10
153	J. W.	24	69	175	94	145	179	+23
154	W. G. L.	23	67	170	93	146	161	+10

TABLE 3.—OBSERVED DATA—*Continued*

Number	Name	Age at Nearest Birthday, in Years	Height Without Shoes		Chest Girth at Nipple, Mean, in Cm.	Actual Weight, Stripped		
			In.	Cm.		July 1918, in Lbs.	Jan., 1919, in Lbs.	Per Cent. Gain (+) or Loss (—)
1	2	3	4	5	6	7	8	9
155	J. J. K.	26	66	168	93	147	148	+ 1
156	H. W. T.	29	71	180	91	147	157	+ 7
157	C. D. K.	24	71	180	91	147	164	+12
158	J. P. G.	29	66	168	91	148	155	+ 5
159	G. S.	23	67	170	91	148	148	0
160	W. D. L.	26	71	180	91	148	149	+ 1
161	P. J. M.	23	67	170	89	148	157	+ 6
162	M. N. K.	23	69	175	86	148	170	+15
163	G. E. K.	30	67	170	98	148	170	+15
164	J. B. R.	24	69	175	88	148	178	+20
165	R. L. P.	25	70	178	91	149	155	+ 4
166	C. W. S.	25	68	173	89	149	162	+ 9
167	W. S.	25	63	160	94	150	159	+ 6
168	J. S.	30	69	175	95	150	168	+12
169	F. S. W.	25	70	178	94	150	157	+ 5
170	D. L. H.	31	71	180	90	150	157	+ 5
171	C. J. M.	31	68	173	95	150	164	+ 9
172	J. S.	28	70	178	85	150	142	— 5
173	H. O. W.	29	67	170	95	150	169	+13
174	S. W.	28	68	173	94	151	168	+11
175	A. J. S.	26	68	173	95	151	177	+17
176	R. T.	22	62	157	92	152	155	+ 2
177	E. P. A.	29	70	178	94	152	162	+ 7
178	C. A. E.	23	70	178	89	152	165	+ 9
179	H. S. F.	29	70	178	91	152	169	+11
180	H. S.	24	68	173	97	152	169	+11
181	E. E. P.	25	70	178	93	153	165	+ 8
182	F. D. S.	25	70	178	91	153	171	+12
183	L. R.	29	66	168	97	154	170	+10
184	C. M.	22	64	163	97	155	157	+ 1
185	M. A. M.	24	74	188	88	155	159	+ 3
186	H. A. T.	25	68	173	89	155	164	+ 6
187	E. H. B.	28	71	180	90	156	157	+ 1
188	D. W. E.	24	71	180	89	156	160	+ 3
189	T. J. C.	31	64	163	95	156	166	+ 6
190	J. H. C.	31	71	180	98	156	170	+ 9
191	F. S. T.	23	69	175	89	156	171	+10
192	E. A. B.	24	69	175	93	156	176	+13
193	R. M.	28	67	170	96	158	146	— 8
194	D. E. R.	28	70	178	86	158	160	+ 1
195	J. B. M.	27	70	178	95	158	161	+ 2
196	W. B. W.	20	68	173	94	158	165	+ 4
197	J. J. K.	29	68	173	94	158	172	+ 9
198	J. B. O.	29	68	173	99	158	174	+10
199	J. C. T.	28	72	183	85	160	153	— 4
200	R. E. S.	23	68	173	95	160	155	— 3
201	O. H. W.	31	66	168	95	160	175	+ 9
202	W. M. G.	27	69	175	95	160	175	+ 9
203	W. S.	24	71	180	95	161	179	+11
204	J. D. G.	23	72	183	94	161	194	+20
205	E. E. C.	23	69	175	101	162	173	+ 7
206	A. S. S.	28	68	173	84	163	152	— 7
207	W. M. B.	27	73	185	93	163	168	+ 3
208	P. S. K.	25	68	173	98	164	170	+ 4
209	V. P. N.	26	74	188	91	164	183	+12
210	G. J. S.	28	68	173	87	165	162	—18
211	S. P. M.	26	69	175	95	166	183	+10
212	W. F. R.	25	71	180	95	167	168	+ 1
213	T. J. D.	29	69	175	91	167	170	+ 2
214	H. C. L.	27	67	170	95	168	177	+ 5
215	C. E. B.	26	67	170	94	168	179	+ 7
216	A. A. R.	24	67	170	96	169	166	— 2
217	J. K. Z.	28	72	183	101	170	186	+ 9
218	S. E. S.	23	71	180	106	171	183	+ 7
219	W. C. R.	29	70	178	102	172	168	— 2
220	M. W. H.	27	69	175	102	173	173	0
221	I. L.	31	65	165	99	174	180	+ 3
222	E. S.	22	68	173	70	175	162	— 7
223	T. J. D.	29	69	175	99	175	176	+ 1
224	A. L. A.	30	72	183	98	175	190	+ 9
225	J. C. M.	25	72	183	95	176	185	+ 5
226	W. F. S.	30	65	165	96	179	184	+ 3
227	F. J. R.	30	71	180	95	180	174	— 3
228	E. E. W.	30	71	180	93	190	185	— 3
229	L. A. T.	24	75	191	102	193	236	+22
	Average	26	67	171	90	141	150	+ 7

medicine in a broad sense are either essentially statistical problems, or their statistical phase is a vitally important one in reaching correct conclusions."

Dreyer,[30] and others,[31] have also recently urged greater care as to the method of collecting and presenting facts.

Prediction Error.—When the weight of an individual is predicted by any method the value may be identical with the observed weight, but in general deviates or errs somewhat from it. This error may be either above or below the actual weight, i. e., either positive or negative in sign; and in consequence it tells us whether the predictions made by a given method are on the whole too large or too small. Since we are in this case testing methods of prediction against actual observation we have taken the differences: calculated weight less actual weight, or PW — W = D. This difference D is then reduced from pounds or kilograms to its percentage of the actual weight, plus or minus, i. e., above or below that actual weight; or $\frac{(PW - W)\ 100}{W} = \frac{100\ D}{W} = \%$ prediction error = E. This percentage prediction error is our common unit on which all further discussion is based hereafter. For example, in Table 4, Bornhardt's method applied to Case 1 gives: PW = 55.9 kg. (123 pounds), W = 47.7 kg. (105 pounds), hence D = 8.2 kg. (18 pounds), from which by substitution in the above formula we find the prediction error is 8.2/47.7 = 17 per cent., or 18/105 = 17 per cent. In other words, this value E will be the same no matter whether our observed data have been recorded in metric or customary units, though ordinarily of course all one's data are recorded in the same unit. The error for each individual has been calculated only to the nearest integral, while the average error for each standard is given to about 1 per cent.

The reasons for this percentile manner of expression are:

1. When comparing the varying errors obtained by applying the same formula to the 229 different subjects, percentage errors are always comparable, whereas kilogram or pound errors are often not comparable owing to the subjects' varying weights.

2. When comparing the average of these 229 errors by one formula with the average of the errors by a second formula on the same 229 cases, percentage errors are always comparable, whereas kilogram or pound errors are often not comparable because the two formulas are expressed in different units: one metric, the second English.

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30. Dreyer, G., and Walker, E. W. A.: Iatro-mathematics, in Contributions to Medical and Biological Research, Dedicated to Sir William Osler, New York, **1**:40, 1919.

31. Editorial, J. A. M. A. **73**:1531 (Nov. 15) 1919; also, Kilgore, E. S.: J. A. M. A. **75**:86 (July 10) 1920.

3. Furthermore, as will be clearer further on, the expression of the errors as percentages automatically determines limits of the class intervals convenient for tabulating the frequency of each of the different sized errors, for reckoning the average of the 229 errors, or their standard-deviation, or their coefficient of variation.

Analysis of the error, to determine the relative precision of prediction by different methods, is carried out by the application of the following criteria or constants:

1. The frequency distribution of error.

2. The average error with regard to sign, i. e., the algebraic mean.

3. The average error without regard to sign, i. e., the arithmetical mean.

4. The standard error, i. e., the square root of the sum of the squares of the errors.

5. The range between the maximum error and the minimum error.

6. The standard deviation of the average error, i. e., the square root of the sum of the squares of the deviations of each of the 229 errors from the average error.

7. The coefficient of variation.

The next step is to summarize the errors in the predictions by each method, as given in Table 4, into a frequency-distribution table, Table 5, in conformity with the dictum: "In all cases of published work the figures of the frequency-distribution should be given; they are absolutely fundamental." [32]

Calculation of the Mean Error.—Method I (Yule [32]): From frequency distribution Table 5.

Let A = an arbitrary value or guess at approximate mean.

Let M = true mean.

Let d = difference between approximate and the true mean = M-A = Sum f. z/N.

Then by substitution of figures from Table 5.

$$d = \frac{\text{Sum}\,(f.z)}{N} = \frac{+453-452}{229} = \frac{+1}{229} = 0.0044 \text{ class-intervals.}$$

Now in making the frequency Table 5, each class-interval was taken as = 1 per cent. error.

$$\therefore d = 0.0044 \text{ per cent.}$$

$$M = A + d = 6 + 0.0044 = 6.0044 \text{ per cent.}$$

Method II (Harris and Benedict): From basal Table 3.

$$M = \frac{\text{Sum E}}{N} = \frac{1374}{229} = 6.00 \text{ per cent.}$$

32. Yule, G. U.: Introduction to the Theory of Statistics, Ed. 5, London, 1919, pp. 79, 112, 133, 141, 144, 153.

TABLE 4.—METHOD OF COMPUTING PREDICTION ERROR; WITH EXAMPLES FROM THE APPLICATION OF BORNHARDT'S STANDARD TO THE SERIES OF 229 MALES

	Case Number	Prediction (Standard: Bornhardt's Formula)			
		Predicted Weight	Difference of Predicted Weight from Actual Weight, Too High (+) Too Low (−)	Percentile Prediction Error, Prediction Error, or Simply Error	Error Squared
		PW	PW − W = D	$\frac{D}{W} = E$	E^2
		Kg. or Lbs.	Kg. or Lbs.	Per Cent. of Actual Weight W	
	1	55.9	+ 8.2	+17	289
	2	127	+20	+19	361
	-- -- --	-- -- -- -- --	-- -- -- -- -- --	-- -- -- -- -- --	-- -- -- --
	229	179	−14	− 7	49
Arithmetic sum				1,374	14,283
Mean error				6.0%	62.37
Root-mean-square-error					7.89%
Range of error			0 to +20 and −64 lbs.	0 to +19 and −36%	0 to 1,296
Standard deviation				5.14%	

TABLE 5.—SUMMARY OF PREDICTION ERRORS IN COLUMN E OF ORIGINAL TABLE 4, TO SHOW FREQUENCY-DISTRIBUTION, AVERAGE ERROR AND STANDARD-DEVIATION. STANDARD: BORNHARDT'S. SERIES: 229 SOLDIERS

1	2	3	4	5	6
Percentage Prediction Error	Frequency	Deviation from Arbitrary Value A	Product Column 2 × Column 3	Deviation Squared	Product Column 2 × Column 5
E	f	z	f.z	z^2	$f.z^2$
0	9	− 6	− 54	36	324
1	33	− 5	−165	25	825
2	27	− 4	−108	16	432
3	25	− 3	− 75	9	225
4	16	− 2	− 32	4	64
5	18	− 1	− 18	1	18
			−452		
6	24	0			
7	8	1	8	1	8
8	11	2	22	4	44
9	5	3	15	9	45
10	7	4	28	16	112
11	12	5	60	25	300
12	7	6	42	36	252
13	6	7	42	49	294
14	4	8	32	64	256
15	5	9	45	81	405
16	1	10	10	100	100
17	4	11	44	121	484
18	3	12	36	144	432
19	3	13	39	169	507
36	1	30	30	900	900
			+453		
Sum	229	....	+1	...	6,027
Mean	...	....	+0.0044	...	26.3

Calculation of the Standard Deviation.

Method I (Yule): From the frequency distribution Table 5.

$$\text{S. D.} = \sqrt{\frac{\text{Sum } f.z^2}{N} - d^2}$$

$$= \sqrt{\frac{6027}{229} - (0.0044)^2}$$

$$= \sqrt{26.3 - 0.000019}$$

$$= \sqrt{26.299981}$$

$$= \sqrt{26.3}$$

$$= 5.13\%$$

Method II (Harris and Benedict[38]): From Tables 3 and 4.

$$\text{S. D.} = \sqrt{\frac{\text{Sum } E^2}{N} - \left(\frac{\text{Sum } E}{N}\right)^2}$$

$$= \sqrt{\frac{\text{Sum } E^2}{N} - (M)^2}$$

$$= \sqrt{\frac{14283}{229} - (6.00)^2}$$

$$= \sqrt{62.37 - 36.00}$$

$$= \sqrt{26.37}$$

$$= 5.14\%$$

Average Error with Regard to Sign, Algebraic Mean.—When a given prediction method gives results which are on the average too high, the mean error with regard to sign will have the positive sign.[5] When it is too low, it will have the negative sign. Dividing the sum of the errors with regard to sign by the total number of individuals in the series in hand we have a measure of the average error in the direction of too high or too low prediction. This value is given in Tables 7, 8, 9. It is liable to give a factitious appearance of accuracy because the plus and minus errors tend to cancel, and the algebraic mean is therefore only mentioned in passing, while great attention is given to the arithmetic mean discussed below. Before passing to that however, another measure of the direction of the error may be presented in Table 6.

TABLE 6.—Prediction Error Too High or Too Low

	Number of Times				Percentage of Times			
	Too Low	Too High	Correct	Total	Too Low	Too High	Correct	Total
Bornhardt's	89	131	9	229	39	57	4	100
Army	82	111	9	202	41	55	4	100
Medico-Actuarial	79	144	5	225	35	63	2	100
Guthrie's	46	175	8	229	20	76	4	100
Broca's	27	201	1	229	12	87	1	100
von Noorden's	4	223	2	229	2	97	1	100

Average Error Without Regard to Sign, Arithmetic Mean.—But the question, however, as to whether a given prediction method yields on the whole too high or too low values is not the only one to be answered. One wishes to know the extent of errors, either above or below the actual weight, in the case of each of the methods used. This

extent may be reckoned (1) by ignoring the signs and simply regarding a difference between observed and predicted body weight as an error of a given magnitude in kilograms, or better as an error of the equivalent percentage of the actual weight; then dividing the sum of these errors by the number of individuals in the series, the formula is: Sum E/N = M. Another method (2) for obtaining the mean, useful as a confirmation of the above briefer method, is seen in Table 5.

This mean error sometimes proves identical for two methods under comparison, so that further analysis by other constants is necessary. Furthermore, these other constants may be of service even in cases where they are not made absolutely necessary by similarity of means. For the arithmetical mean has two disadvantages.[8] First, it does violence to sound mathematical usage with regard to signs. Second, it gives errors importance only proportional to their magnitudes, while one may consider, in a comparative test like the present, that very great errors should be given proportionally more weight than very slight deviations. Both of these drawbacks may be avoided by figuring the standard error.

Standard Error, or Root-Mean-Square Error.—In this paper this term is used in another sense than that usual among statisticians. Here, the above mentioned magnitudes of the deviations or of the percent errors may be logically weighted and the transgression against the law of signs avoided by squaring the errors, summing the squares, obtaining their mean and extracting its squares.

Range.—The error made by Bornhardt's or any other method is of course variable, and for this variation, technically called dispersion, "the simplest possible measure . . . is the actual *range*, i. e., the difference between the greatest and least values observed. While this is frequently quoted, it is as a rule the worst of all possible measures for any serious purpose." [32] A better measure is the standard deviation, and better still, the coefficient of variation.

Standard Deviation.—Yule [32] states that "The standard deviation should always be used as the measure of dispersion, unless there is some very definite reason for preferring another measure, just as the arithmetic mean should be used as the measure of position." The standard deviation is the square root of the average of the squares of all deviations as measured from the arithmetic mean of the observations; and therefore is called the root-mean-square deviation from the mean. To square all the deviations may seem artificial, but such root-mean-square quantities frequently occur in other branches of science. The computation may be made either from the frequency Table 5, modeled on Yule, or from the briefer formula of Harris applied to figures in Table 4.[8, 33]

33. Harris, J. A.: Am. Naturalist **44**:693, 1910.

Coefficient of Variation.—Standard deviations are not comparable when the respective means are of different size, hence the need of translating the former from absolute to percentage values, thus securing the coefficients of variation. The formula is 100 S.D./M. = C. V.

DATA COMPUTED AND ANALYZED

Error in Relation to the Six Standards.—Our findings are summarized for the first series in Table 7, for the second or present group in Table 8, and for the two groups consolidated in Table 9. In each table note the lower values across the first row, as compared with successive rows down the page.

TABLE 7.—ERROR IN RELATION TO STANDARDS. SUMMARY OF PREDICTION ERRORS COMPUTED FROM ORIGINAL OF TABLE 4. STANDARDS: ALL SIX. SERIES: TWENTY MALES

Rank in Order of Accuracy	Standards Compared	Number of Subjects	Average or Mean Error: Sum E / N — Algebraic Mean: Average With Regard to Sign	Average or Mean Error: Sum E / N — Arithmetic Mean: Average Without Regard to Sign	Root-Mean-Square Error or Standard Error: $\sqrt{\frac{\text{Sum } (E^2)}{N}}$	Standard-Deviation of Mean Error: $\sqrt{\frac{\text{Sum } (E^2)}{N} - M^2}$	Coefficient of Variation of Mean Error: $\frac{100\ SD}{M}$
		N	A	M	S.E.	S.D.	C.V.
1	2	3	4	5	6	7	8
1	Bornhardt's	20	− 2.7	5.6 ± 0.542	6.6	3.6	64.3
2	Army	18	+ 2.6	7.3 ± 1.03	9.8	6.5	89.0
3	Medico-Actuarial	20	+ 3.1	7.4 ± 0.77	8.9	5.1	68.9
4	Guthrie's	20	+ 8.8	9.5 ± 1.22	12.5	8.1	85.3
5	Broca's	20	+13.1	13.1 ± 1.37	15.9	9.1	69.5
6	v. Noorden's	20	+19.8	19.9 ± 1.84	23.4	12.2	61.3

TABLE 8.—ERROR IN RELATION TO STANDARDS. STANDARDS: ALL SIX. SERIES: 229 SOLDIERS

Rank in Order of Merit	Standards Compared	Number of Cases	Average or Mean Error		Standard Error	Standard Deviation	Coefficient of Variation
		N	A	M	S.E.	S.D.	C.V.
1	Bornhardt's	229	+ 1.4	6.0 ± 0.228	7.9	5.1	85.0
2	Army	202	+ 2.0	8.4 ± 0.318	10.7	6.7	79.6
3	Medico-Actuarial	228	+ 3.5	8.7 ± 0.291	10.9	6.5	74.7
4	Guthrie's	229	+ 8.7	11.7 ± 0.374	14.4	8.4	71.8
5	Broca's	229	+13.0	14.7 ± 0.418	17.4	9.4	64.0
6	v. Noorden's	229	+23.6	23.7 ± 0.504	26.3	11.3	47.7

Error in Relation to Age.—The question may be raised, do prediction errors vary proportionately with age, actual weight, height, or chest-girth (either directly, inversely, or at the extremes from the average)? The age ranged from 18 to 34 years, with an average of

25.9 years. The mode, i. e., the most frequent or typical age, was 23 years. Eighty-six per cent. of the men were in their twenties. Ninety per cent. were 23 or over, "the age when the average man has attained his full stature and bulk," according to Quetelet,[6] Dawson, Aitkin and Beddoe.[21] It should be remembered that this age is taken from the nearest, not the last, birthday. The frequency distribution shows that the error is least at the average age, i. e., 26, runs high at isolated ages, but does not tend to be greater over the younger or indeed over any significant period. The errors for the extreme age groups are bracketed, as being unreliable, owing to the small number of cases in each of those classes (Table 10).

TABLE 9.—Error in Relation to Standards.
Series: 20 + 229 = 249 Males

Rank in Order of Merit	Standards Compared	Number of Subjects	Average or Mean Error		Standard Error	Standard Deviation	Coefficient of Variation
		N	A	M	S.E.	S.D.	C.V.
1	Bornhardt's	249	+ 1.1	6.0 ± 0.213	7.8	5.0	83.3
2	Army	220	+ 2.0	8.3 ± 0.304	10.7	6.7	80.7
3	Medico-Actuarial	248	+ 3.5	8.6 ± 0.274	10.7	6.4	74.4
4	Guthrie's	249	+ 8.7	11.5 ± 0.358	14.3	8.4	73.0
5	Broca's	249	+13.0	14.6 ± 0.396	17.3	9.3	63.7
6	v. Noorden's	249	+23.2	23.4 ± 0.485	26.0	11.4	43.7

TABLE 10.—Distribution of Error According to Age
Standard: Bornhardt's. Series: 229 Males

	Nearest Age																	
	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	Sum
Class-frequencies, i. e., number of cases in each class	1	1	1	5	15	38	22	31	19	20	30	15	12	12	2	3	1	229
Average error at each age	(2	11	5	6)	9	5	5	6	4	6	7	5	9	7	(6	5	11)	

Error in Relation to Actual Weight.—The weight varied from 47.6-87.5 kg. (105-193 lbs.), with a mean of 64.1 kg. (141 lbs.), and a mode of 61.2 kg. (135 lbs.). The frequency distribution is summarized in Table 11. In choosing the magnitude of the class-interval, the frequency distribution unit of 1 pound each would have given 88 classes which would have been unwieldy and would have included too few figures in each class; units of one kilogram each with 40 classes, would have the same drawbacks; 2 kilogram intervals would have given the very desirable number of classes,[22] but would still have had a rather small number of cases in each. The size chosen also has the advantage of keeping both kilos and pounds as integers. Even with only eight classes the last covers only two cases, and must be ignored in the inter-

pretation. The error is seen to be least for the groups including the central weights, as would be expected, and to increase toward the extremes.

Error in Relation to Height.—The stature in bare feet averaged 171 cm. (67 inches), ranging from 147-191 cm. (58-75 inches). No correlation is seen in Table 12.

TABLE 11.—DISTRIBUTION OF ERROR ACCORDING TO WEIGHT *
STANDARD: BORNHARDT'S. SERIES: 229 MALES

Weight										
	Kg.	48 and under 53	53—	58—	63—	68—	73—	78—	83—	47.6-87.5
	Lbs.	106 up	117—	128—	139—	150—	161—	172—	183—	105-193
Number of cases		18	31	73	49	36	16	9	2	229
Average error for each weight class		12	9	5	4	5	7	13	(13)	

* Each class-interval = 5 kg. = 11 lbs.

TABLE 12.—DISTRIBUTION OF ERROR ACCORDING TO HEIGHT *
STANDARD: BORNHARDT'S. SERIES: 229 MALES

Height										
	Cm.	147 and under 152	152—	157—	162—	167—	172—	177—	182—	187—	147-191
	In.	58 up	60—	62—	64—	66—	68—	70—	72—	74—	58-75
No. of cases		1	2	19	33	51	65	43	11	4	229
Average error for each class		(2 | 11)	5	8	5	6	5	6	(7)	

* Each class interval = 5 cm. = 2 inches.

TABLE 13.—DISTRIBUTION OF ERROR ACCORDING TO CHEST-GIRTH *
STANDARD: BORNHARDT'S. SERIES: 229 SOLDIERS

Chest girth													
	Cm.	70	79 and under 81.5	81.5—	84—	86.5—	89—	91.5—	94—	96.5—	99—	101.5—	104—	70-106
	In.	28	31 up	32—	33—	34—	35—	36—	37—	38—	39—	40—	41—	28-42
No. of cases		1	4	9	40	29	79	15	33	9	6	3	1	229
Average error for each class		36	3	8	6	5	6	7	6	5	4	5	2	

* Class interval = 2.5 cm. = 1 inch.

Error in Relation to Chest-Girth.—The perimeter of the thorax, at the level of the mammillae, midway between inspiration and expiration, varied from 70 to 106 cm. (from 28 to 42 inches) with an average of 90 cm. (35 inches). No correlation is evident (Table 13).

CONCLUSIONS

The normal weight for an American man may be predicted somewhat more accurately by Bornhardt's formula than by the Army and Navy table, the Medico-Actuarial, Guthrie's, Broca's, or Von Noorden's standard. This was true for a series of 249 native-born men, aged 18 to 34 years, inclusive. Whether it would also hold true for other men, for women and for children, we cannot state. Bornhardt's standard did not vary in accuracy at varying ages, weights, heights and chest-girths. Though other writers have urged the value of routinely recording the chest perimeter as well as height, nobody has incorporated it into a formula as simple as Bornhardt's. And, just as his rule (based on height and chest-girth) as a rough measure of surface seems both theoretically and empirically the best guide so far offered for guessing weight, so we expect that experiment would discover an even better rule (based on height, chest and a third factor) expressing a rough measure of volume. For fundamentally weight must be proportional, not to length nor surface, but to cubic mass.

We wish to express our thanks for assistance of various kinds to Prof. I. I. Lemann of New Orleans, formerly Major M. C. and chief of the medical service of the unit, to Dr. A. B. Brower of Dayton, Ohio, to Dr. F. B. Allen of North Wales, Pa., and to Prof. J. L. Coolidge of Cambridge, Mass.

BUNDLE BRANCH BLOCK AND ARBORIZATION BLOCK *

FRANK N. WILSON, M.D., AND GEORGE R. HERRMANN, M.D.
ST. LOUIS

For the first few years after the introduction of the string galvanometer into clinical medicine, electrocardiographers devoted most of their attention to the analysis of the cardiac irregularies. This field has been so extensively cultivated that it is approaching exhaustion, and there is an increasing tendency to turn to the significance of abnormalities of the form of the electrocardiographic deflections as a subject of research. The notable contributions which have recently been made to this subject, and the confusion that must result from the conflicting views that have lately been expressed, make it seem advisable to sum up, in a critical way, the present state of our knowledge. The present article is devoted to a careful digest of the literature on those abnormalities of the ventricular complex which indicate disturbances of intraventricular conductivity, and to the presentation of a number of original observations which have a bearing on their interpretation.

EARLY WORK ON BUNDLE BRANCH BLOCK

Our knowledge of the changes in the form of the ventricular complex which result from lesions of the two chief divisions of the His bundle dates from the work of Eppinger and Rothberger.[1] These authors made a large number of experiments on dogs in which they injected solutions of silver nitrate into the ventricular muscle with the idea of determining the effect of destroying various portions of the muscle of these chambers on the electrocardiogram. They found that in many instances a comparatively large mass of muscle might be injured without changing the form of the electrocardiogram, while in other experiments relatively small lesions produced very striking results. They were struck by the effectiveness of injections into the interventricular septum in comparison with those made into the free walls of the ventricles, and they suspected that this was due to injuries of the larger branches of the conducting system which lie beneath the septal endocardium. In a second series of experiments [2] they confined

* From the Department of Internal Medicine, Washington University Medical School.

1. Eppinger, H., and Rothberger, J.: Zur Analyse des Elektrokardiograms, Wien. klin. Wchnschr. **22**:1091, 1909.

2. Eppinger, H., and Rothberger, J.: Ueber die Folgen der Durchschneidung der Tawaraschen Schenkel des Reizleitungsystems, Ztschr. f. klin. Med. **70**:1, 1910.

their attention to these special tissues, and by introducing a small knife through the ventricular wall they were able to sever the chief right and left branches of the His bundle. Remarkable changes in the form of the electrocardiogram resulted. A single lead from esophagus to rectum was used. When the right branch was cut, the normal, relatively small, physiologic ventricular complexes gave place to diphasic complexes of large amplitude closely resembling the ventricular complexes obtained by electrical stimulation of the surface of the left ventricle. The initial phase (QRS) was downwardly directed and was of greatly increased duration; it was immediately followed by a large upwardly directed final deflection, an exaggerated T. Section of the left branch produced similar results, except that the direction of both phases was reversed so that the complexes resembled those obtained by stimulation of the surface of the right ventricle.

While these experiments were in progress, the authors were on the lookout for clinical cases showing electrocardiographic abnormalities similar to those that they had produced experimentally, and Eppinger and Stoerk [3] were fortunate in discovering five such cases. Two of these patients died while in hospital and necropsies were obtained. Both had been observed over long periods and had constantly shown abnormal ventricular complexes of a definite type. These complexes were typically diphasic in all leads; both phases were of large amplitude in Leads I and III, and the QRS interval was much greater than normal. The first phase (QRS) was upwardly directed in Lead I and downwardly directed in Leads II and III. Since Leads II and III corresponded more closely to the esophageal-anal lead used in the previously mentioned experiments than did Lead I, the authors made the diagnosis of right bundle branch block in each case. At the necropsies very careful macroscopic and microscopic examinations of the branches of the His bundle were made and in both instances lesions were found completely transecting the right branch while the left branch was normal. These pathologic findings are of the greatest importance for, so far as we know, they are the only ones of their kind on record. They demonstrate that lesions that transect the right branch of the His bundle in man produce diphasic ventricular complexes of which the first phase (QRS) is upwardly directed in Lead I, and downwardly directed in Lead III. The conclusion arrived at by Fahr [4] from theoretic considerations that what we are accustomed to call right bundle branch block is really left bundle branch block is not in accord with these facts.

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3. Eppinger, H., and Stoerk, O.: Zur Klinik des Elektrokardiograms, Ztschr. f. klin. Med. **71**:157, 1910.

4. Fahr, G.: An Analysis of the Spread of the Excitation Wave in the Human Ventricle, Arch. Int. Med. **25**:146 (Feb.) 1920.

The criteria to be used in recognizing bundle branch block were not very clearly stated by Eppinger and his collaborators. Carter,[5] working in Lewis' laboratory, published a large series of curves in 1911 which he interpreted as examples of the bundle branch block, and gave a table contrasting the features of the normal and the abberrant ventricular complex.

Normal	Abberrant
1. Supraventricular complex. Presence of auricular or P summits.	1. Supraventricular complex. Presence of auricular or P summits.
2. P-R interval 0.13 to 0.18 second, never more than 0.2 second.	2. P-R interval frequently prolonged beyond 0.2 second.
3. QRS interval less than 0.1 second and less than one-third of entire complex.	3. QRS interval exceeds 0.1 second and as a rule constitutes more than one-third of entire complex.
4. Relatively small amplitude of initial deflections.	4. Relatively increased amplitude of initial deflections.
5. Final deflection T upright and in the same direction as the most prominent deflection (R) in Leads I and II, and usually in Lead III.	5. Final deflection T′ usually in a direction opposite to that of the prominent initial deflection.
6. Initial deflections, as a rule unnotched.	6. Initial deflections almost always show notching in one lead at least. Many bizarre forms seen.
7. Final deflection T as a rule plainly to be seen, but not exaggerated.	7. Final deflection T′ frequently much exaggerated.

Not all of the curves published by Carter as examples of bundle branch block show all the abnormalities catalogued in the second column of the table; many are not of particularly large amplitude; others do not show a QRS interval greater than 0.1 second. All, however, are diphasic in Leads I and III. Two cases, one in which the abberrant electrocardiograms were transitory and one in which they developed while the patient was under observation, are of unusual interest. The first illustrates the contrast between curves indicating left ventricular preponderance and those indicating right bundle branch block and the second illustrates the contrast between the abberrant curves and electrocardiograms of relatively normal outline from the same individual. In none of Carter's cases were necropsies reported.

Cohn and Lewis,[6] however, reported the pathologic findings in four cases of supposed bundle branch block from the same laboratory. In none of these cases could the electrocardiographic diagnosis be substantiated. In commenting on the discrepancy between the electrocardiographic and pathologic findings, the authors point out that the functional changes shown by electrocardiograms do not necessarily rest on recognizable pathologic lesions.

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5. Clinical Observations on Defective Conduction in the Branches of the A-V Bundle, Arch. Int. Med. **13**:803 (July) 1914.

6. Cohn, A., and Lewis, T.: The Pathology of Bundle Branch Lesions of the Heart, Proc. New York Path. Soc. **14**:207, 1914.

Further clinical examples of bundle branch block have been reported by Mathewson [7] and others; frequent examples are seen at every clinic where large numbers of patients with heart disease are studied electrocardiographically. It is notable that right bundle branch block is very much more common than left bundle branch block, and this has been attributed to the difference in the character of the two branches. The right branch passes to the chief papillary muscle of the right ventricle as a single strand while the left branch subdivides almost at once spreading out into a broad fan so that it can hardly be completely divided, except by a relatively large lesion.

LATER WORK ON BUNDLE BRANCH BLOCK

Following certain criticisms of the work of Eppinger and his collaborators, Rothberger and Winterberg [8] repeated the experimental work of Eppinger and Rothberger, using in this instance the ordinary three leads. They found that when one of the chief branches of the His bundle was severed, typical diphasic complexes resulted, but the initial deflections (QRS) in Leads I and III had the same and not opposite directions as in the human cases reported by Eppinger and Stoerk.

In the course of his work on the spread of the excitatory process over the heart, Lewis [9] found it necessary to reinvestigate this subject. He found that in the majority of dogs the initial phases of the curves obtained by section of the right branch were downwardly directed in all leads. He termed curves of this type concordant. In a small percentage of dogs QRS of Lead I was upwardly directed as in right bundle branch block in man. These curves were termed discordant. Section of the left branch always gave curves of the concordant type; QRS was upwardly directed and T downwardly directed in all leads. Why should right bundle branch block produce concordant curves in most dogs and discordant curves in man? Lewis gives evidence to show that it is not due to a difference in the position of the heart with reference to the leads used nor to other similar factors. The discordant curves obtained from a small percentage of dogs furnish the solution; the cause is to be found in the heart itself. Lewis observed that he could predict from the form of the physiologic electrocardio-

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7. Mathewson, G. D.: Lesions of the Branches of the Auriculo-Ventricular Bundle, Heart **4**:385, 1913.

8. Rothberger, C. J., and Winterberg, H.: Zur Diagnose der einseitigen Blockierung in den Tawaraschen Schenkeln, Zentralbl. f. Herz u. Gefässkr. **5**:206, 1913.

9. Lewis, T.: The Spread of the Excitatory Process in the Vertebrate Heart, Phil. Tr. Roy. Soc. Lond. **207**:221, 1916.

gram of a given animal which type of curve would result from section of the right branch of the bundle. When R1 (R of Lead I) was of small amplitude, concordant curves resulted, and when R1 was of large amplitude, discordant curves were produced. An inspection of the hearts from which discordant curves were obtained showed that there was less bridging of the cavity of the left ventricle by subdivisions of the left branch than in hearts which gave concordant curves. Lewis, therefore, attributes the difference between the curves of bundle branch block in the dog and the curves of similar lesions in man to greater bridging of the ventricular cavities in the former by strands of special conducting tissue. The experiment performed by Lewis on a Rhesus monkey indicates that in this animal the curves of bundle branch block are of the discordant type, as in man.

ANALYSIS OF THE FORM OF THE VENTRICULAR COMPLEX IN BUNDLE BRANCH BLOCK

The analysis of the ventricular complex in bundle branch block and the analysis of the normal ventricular complex are very closely related and will be discussed together. In each case the analysis depends on a knowledge of the course of the excitation wave over the ventricular muscle. From the auricles the normal impulse passes down the His bundle and its chief branches, through the larger and smaller subdivisions of these branches, into the Purkinje network which lines both ventricles.[10] From the Purkinje network, which does not penetrate the ordinary muscle deeply, the impulse passes into the muscle of the ventricular walls. The rate of travel through the Purkinje tissue is about ten times the rate through the ordinary ventricular muscle (the figures are, from 3,000 to 5,000 mm. per second for the former and from 300 to 500 mm. per second for the latter). As a result of its much greater speed of travel through the conducting system, and the failure of this system to penetrate the ordinary muscle deeply, the excitation wave spreads over the endocardial surface very quickly and its average course through the ventricular walls is roughly perpendicular to the endocardial surface (Fig. 1). When, as in man, there is little or no bridging of the ventricular cavity by special conducting tissue, the excitation wave spreads down the Purkinje network of the septum toward the apex and thence upward through the network of the free walls; the subendocardial muscle of these regions is activated in the same order. Because of its relatively slow rate of progress through the ordinary muscle, however, the time of arrival of the excitatory process at a point on the epicardial surface of the ventricles is largely

10. Lewis, T., and Rothschild, M. A.: The Excitatory Process in the Dog's Heart. Part II, The Ventricles, Phil. Tr. Roy. Soc. Lond. **206**:181, 1915.

controlled by the thickness of the muscle at that point. The course of the excitation wave over the ventricular muscle is diagrammatically shown in Figure 1, which also shows the relationship of the direction in which various regions are activated to the equilater l triangle of Einthoven. It was shown by Lewis and his collaborators that the time taken by the impulse to complete its course through the ventricular muscle was equal to the QRS interval and that the deflections which comprise the QRS group are produced by the activation of the ventricles.

Fig. 1.—Diagrammatic representation of the course of the excitation wave over the ventricular muscle. The direction of the excitation wave through the ventricular walls is shown by arrows. The relationship of the course of the excitation wave through the ventricular walls to the equilateral triangle of Einthoven is shown. e1 equals deflection in Lead I, e2 equals deflection in Lead II, e3 equals deflection in Lead III. See text and compare with Figure 6.

It was shown also [10] that section of the right branch of the His bundle caused a great delay in the spread of the excitation wave over the muscle of the right ventricle; the activation of the left ventricle was not disturbed. This delay is due to the absence of any connecting link between the conducting networks of the two ventricles below the main stem of the His bundle, so that when the right branch is cut, the excitatory impulse can only reach the right ventricular Purkinje system

by passing through the ordinary muscle of the ventricular septum. It is mainly the slow speed of travel through the septum that causes the great QRS interval of bundle branch block. Since section of the right branch of the His bundle delays the activation of the right ventricle but does not disturb the activation of the left ventricle, the first part of the QRS group of right bundle branch block curves is an expression of pure left ventricular effects (levogram). Similarly, the first part of the QRS group of left bundle block curves is an expression of pure right ventricular effects (dextrogram). By clamping first the right and then the left branch in a dog Lewis [9] was able to obtain both levogram and dextrogram, as well as the physiologic electrocardiogram, from the same animal. Synchronous points on these curves were obtained by a method which need not be described here. It was then found that by algebraic summation of the levogram and dextrogram the normal QRS group could be reproduced. The extraordinarily close resemblance of the calculated to the recorded normal QRS in this experiment proves that the normal QRS group is a summation of right and left ventricular effects, and that these effects can be obtained separately by producing bundle branch block first on one side and then on the other.

In the monkey and in man the dextrogram usually begins with a slight upward deflection (R′) in Lead I and a small downward deflection (Q′) in Lead III. The main deflection is downwardly directed (S′) in Lead I and upwardly directed (R′) in Lead III. The levogram is similar in form, but the main deflection is upward in Lead I (R′) and downward in Lead III (S′), and the preliminary deflections (Q′1 and R′3) are also oppositely directed to the preliminary deflections of the dextrogram in the same leads. It is apparent, therefore, that the constitution of the normal QRS group of man is as follows; Q 1 (Q of Lead I) is a left, and Q 3 a right ventricular effect; R 1 is mainly a left, and R 3 mainly a right ventricular effect; S 1 is a right, and S 3 a left ventricular effect (Lewis [9]).

ELECTRICAL EFFECTS PRODUCED BY THE ACTIVATION OF A SIMPLE MUSCLE AND THEIR RELATIONSHIP TO THE INTERPRETATION OF THE NORMAL AND ABNORMAL VENTRICULAR COMPLEX

From a theoretical consideration of the electrical effects produced by the activation of simple muscles, Fahr [4] reached conclusions which are not in accord with Lewis' analysis of the normal electrocardiogram, to which we have adhered in this paper, and which have led him to believe that the abnormal ventricular complexes which are at present regarded as evidence of right bundle branch block are really due to

left bundle branch block. Before proceeding with the analysis of the curves of bundle branch block, it is necessary to examine this subject in some detail.

Let A-B (Fig. 2) represent a simple strip of heart muscle immersed in a conducting medium to which the galvanometer terminals are attached in such a way that relative negativity at R will cause an upward and relative negativity at S a downward deflection in the completed electrogram.

The muscle A-B is asymetrically placed with reference to the ends of the container R and S at which the electrodes are attached. Will the asymmetric position of the muscle influence the form of the elec-

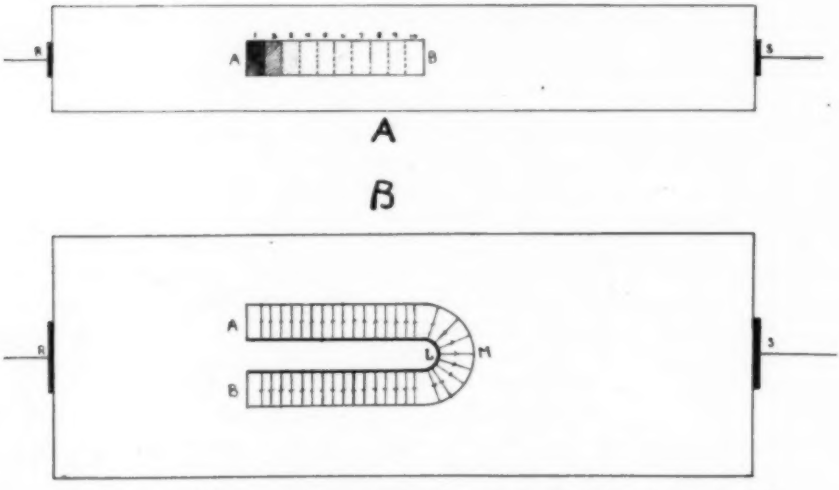

Fig. 2.—A, Diagrammatic representation of simple strip of muscle A-B immersed in conducting medium with electrodes attached at R and S. (see text). B, diagrammatic representation of horseshoe shaped piece of muscle immersed in conducting medium with electrodes attached at R and S. Muscle lined by Purkinje tissue (heavy line). Arrows represent course of excitation wave through muscle when point on internal surface is stimulated.

trogram obtained when the muscle contracts? From a consideration of the distribution of the iso-potential surfaces throughout a conductor when a difference of potential is generated within it, it appears that the asymmetric position of the muscle will not influence the direction of the deflections produced.[11] It may also be stated that it will not influence the amplitude of these deflections, providing that the distances R-A and B-S are relatively large in comparison with the distance A-B and the magnitude of the potential produced by the muscle.

11. Private Letter, Einthoven to Lewis, 1918.

The truth of the last statement is shown by the following experiment. A series of electrocardiograms were taken from a normal subject in the manner illustrated by Figure 3. A line was drawn from the fourth costal cartilage (A) to a point on the left leg just below Poupart's ligament (E). This line was divided into four equal parts, 5 inches in length (A-B, B-C, C-D, D-E). By connecting in succession each two adjacent points to the terminals of the galvanometer four tracings were obtained (Fig. 4). It will be seen that the amplitude of the deflections falls off very rapidly in passing from lead A-B to lead

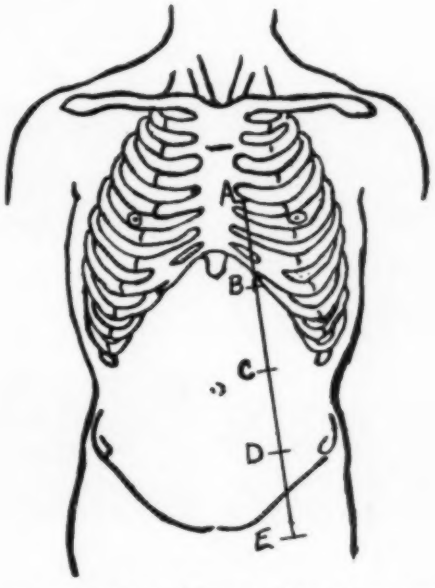

Fig. 3.—Diagram illustrating manner of taking electrocardiograms shown in Figure 4. A, fourth costal cartilage. Distances A-B, B-C, C-D and D-E equal and 5 inches in length.

C-D, and that no curve was obtained from lead D-E; that is, points D and E were of the same potential. The change in potential per unit distance decreases very rapidly, therefore, as the distance from the source of potential difference increases. We conclude that the asymmetric position of the muscle A-B with reference to the electrodes at R and S is of no importance so long as the distances R-A and B-S are relatively great.[12]

12. In comparison with the distance A-B and the magnitude of the potential produced by the muscle.

It is not our purpose to discuss the form of the electrogram that might be expected to result from the activation of the muscle A-B. It is clear that if the muscle be stimulated at A so that the excitation wave travels from A to B, the first deflection that results will be directed upward. If the muscle is stimulated at B so that the excitation wave travels from B to A the initial deflection will be directed downward. But the exact form of the curve can only be determined by experiment; it depends on many factors, and among these the length of the muscle, the speed with which the excitation wave travels, and the form of the curve representing the development, duration and decline of relative negativity in a given section of the muscle. It is obvious that our present knowledge is insufficient to determine the

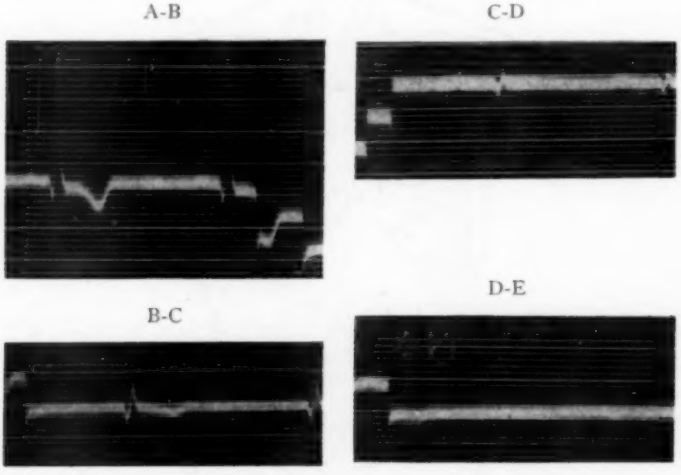

Fig. 4.—Electrocardiograms obtained by connecting successive points of Figure 3. Lead A-B by connecting points A and B to terminals of galvanometer, etc.

exact form of the electrogram produced by the activation of a given muscle, except by experiment. We can, however, predict the direction of the first deflection of this electrogram *providing we know the point of primary negativity and the course of the excitation wave through the muscle.*

Let us now direct our attention to a slightly more complicated muscle. Figure 2B represents a simple strip of heart muscle bent into the shape of a horseshoe and immersed in a conducting medium with electrodes attached as before. If we stimulate this muscle at A or at B so that the excitation wave passes from A or B toward M, the first deflection will be upward. If we stimulate the muscle at M so that the excitation wave travels from M toward A and B the first

deflection will be downward. Can we conclude that an upward deflection indicates primary or preponderant activity at the base of the horseshoe (A or B), and that a downward deflection indicates similar activity at the apex of the horseshoe (M or L) if the point stimulated and the course of the excitation wave over the muscle is unknown? Suppose that the horseshoe is lined with Purkinje tissue which conducts the impulse at least ten times as rapidly as the remainder of the muscle. If any point on the inner surface of the horseshoe is stimulated, the course of the excitation wave over the horseshoe will then be nearly that represented by the arrows. The course of the excitation wave through the limbs of the horseshoe and the direction of the potential differences produced by the activation of these portions of the muscle will be roughly perpendicular to the line of lead and will have little or no effect on the electrogram. But the potential differences produced by the activation of the curved piece of muscle at the apex of the horseshoe will not be perpendicular to the line of lead; the internal surface (L) will show primary negativity with reference to the external surface (M). Since the slightly asymmetric position of the apical muscle with reference to the electrodes (R and S) will not affect the form of the electrogram produced, an upward deflection will result. If, however, the special conducting tissue lay on the external surface of the horseshoe instead of on the internal surface, the external surface (M) would show primary activity with reference to the internal surface (L) and a downward deflection would result. The effect of muscle activity at the apex of the horseshoe depends, therefore, on the course of the excitation wave over the muscle and not altogether on the position of the apex with reference to the remainder of the muscle.

To assume that preponderant muscle activity at the apex of the heart must cause a downward deflection in Lead II, or that corresponding activity at the base must cause an upward deflection in the same lead as has so frequently been done; to assume, as Fahr [4] has done, that when the electrical axis as determined by Einthoven's formula points to the left the preponderant muscle activity is on the right side of the heart, is totally unjustifiable. The facts, so far as they have been determined, indicate that the direction of the electrical axis at any instant corresponds in direction to what may be termed the average direction of the excitation wave at that instant. For example; the excitation wave passes from the endocardial to the epicardial surface of the free wall of the left ventricle; its average direction at this time is upward and to the left. The difference in potential produced by the activation of this region and the corresponding electrical axis is likewise directed upward and to the left; and the resulting deflection is upward in Lead I and downward in Lead III. When several areas

of muscle are passing into the excited state at the same time, the actual potential difference produced is the resultant of the potential differences produced in the various active areas and the electrical axis has the direction of this resultant. The direction of the potential difference produced by the activation of any portion of the ventricular muscle when the heart is activated in the normal way may roughly be determined by means of the scheme shown in Figure 1.

THE ELECTRICAL AXIS IN BUNDLE BRANCH BLOCK

Lewis [9] found on the analysis of the human dextrogram that the electrical axis was at first directed upward and to the left corresponding to the early spread of the excitation through the upper septum in this direction (Fig. 1); later it was directed downward and to the left as the lower portions of the septum became active. Then the electrical axis gradually rotated in a clockwise direction as the free wall of the right ventricle passed into the active state. These changes in the direction of the electrical axis are due to corresponding changes in the direction taken by the excitation wave (Fig. 1). An analysis of the human levogram showed a counter clockwise rotation of the electrical axis corresponding to successive involvement of the septum, apex and free wall (Fig. 1). Since the normal QRS group is a summation of levogram and dextrogram, it appears that Q 1 (Q of Lead I) is produced by the activation of the upper septum from the left side; that Q 3 is produced by the activation of the upper septum from the right side; that R 1 and R 3 are produced by the activation of the lower septum and adjacent portions of the apices of the two ventricles, and that S 1 is produced by the activation of the free wall of the right and S 3 by the activation of the free wall of the left ventricle.[9]

THE CRITERIA USED IN THE RECOGNITION OF BRANCH BUNDLE BLOCK

Let us now take up Carter's criteria for the recognition of bundle branch block in more detail. We shall speak mainly of right bundle branch block, as left branch block is a very rare condition in man.

The P-R Interval.—It is obvious that a lengthened P-R interval gives no direct evidence of bundle branch block. It indicates delayed conduction of the impulse through the A-V node or through the main stem of the His bundle. It is an aid in recognizing bundle branch block only because it indicates a lesion of the special conducting system, and because a lesion of one part of this system is apt to be accompanied by lesions of neighboring parts. No increase in the P-R interval occurs when bundle branch block is produced in animals by cutting the branches of the His bundle.

The QRS Interval.—It has been said that the increased duration of the QRS group in bundle branch block is mainly due to the slow spread of the excitation wave through the septum. The QRS interval will depend, therefore, on the thickness of the septum; it will probably be greater when there is great cardiac hypertrophy than when the heart is of more normal size. It is in patients with great cardiac hypertrophy that bundle branch block is most common, and in our experience the QRS interval of bundle branch block curves is usually much more than 0.1 second, and often more than 0.15 second. In experimental bundle branch block the QRS interval is about twice as great as the QRS interval of the physiologic electrocardiogram. In using the QRS interval as a criterion in recognizing bundle branch block, it must be remembered that as pointed out by Lewis [9] the QRS interval of left ventricular preponderance curves is often more than 0.1 second. This is due to the increased time taken by the excitation wave to traverse the thickened walls of the left ventricle. We believe that an increased QRS interval is the most reliable sign of bundle branch block; the greater the interval the greater its reliability.

Increased Amplitude of QRS.—The large amplitude of the QRS group in bundle branch block is mainly due to a lack of balance. Normally, the effects produced by the activation of the free wall of the left ventricle and those produced by the activation of the free wall of the right ventricle, which are oppositely directed (Fig. 1), come at the same time and tend to neutralize each other. In bundle branch block these two regions are not activated at the same time, so that the normal balance between them is disturbed and comparatively large deflections result. The actual amplitude of these deflections will depend on the amplitude of the levogram or dextrogram, whichever precedes, and the manner in which this is affected by the abnormal spread of the impulse in the ventricle whose activation is delayed. The amplitude of QRS is of less value than the QRS interval in recognizing bundle branch block. In dogs the amplitude of the initial deflection of right bundle branch block complexes is often less than the amplitude of the initial deflection of the physiologic electrocardiogram of the same animal.

The Exaggerated T.—The cause of the exaggerated T which is oppositely directed to the main deflection of the QRS group in bundle branch block curves is not well understood. It is believed that T represents the decline of the excited state in the ventricular muscle; just as the passage of a muscle from the inactive to the active state is accompanied by a change in its electrical state, so must the return of the muscle from activity to inactivity be accompanied by an electrical change of opposite sign. If each portion of the ventricular muscle

remained in the active state exactly the same length of time, and if deactivation took place as rapidly as activation, T should duplicate QRS in form, but should be oppositely directed. It seems probable that since T is several times as broad as QRS the deactivation process takes place very slowly in comparison with activation, and this may explain the difference in the form of the two deflections. The above ideas of the T deflection rest almost entirely on inference, for there is comparatively little experimental evidence bearing on the problem. We have recently carried out some experiments, however, which indicate that they are in the main correct.

Right branch block was produced in dogs by cutting the right branch of the His bundle. Various points on the surface of the right and left ventricles were then stimulated, sometimes at irregular, sometimes at regular intervals, by single induction shocks of the same strength. Each shock produced a slight notch in the electrocardiogram which could be used as a signal, and sufficient curves were taken to determine the effect of stimuli which fell in the different portions of the cardiac cycle. It was found that, in general, points on the surface of the left ventricle passed out of the refractory state from 0.02 to 0.03 second earlier than points on the surface of the right ventricle (Fig. 5). On the other hand, when left bundle branch block was produced the refractory period persisted longer over the surface of the left ventricle than over the surface of the right. These observations indicate that the ventricle that is first to be activated, first passes out of the active state. We believe that the large positive T of right bundle branch block (Lead II) in dogs is due to the early deactivation of the left ventricle in comparison with the right; and that the large negative T of left bundle branch block is due to early deactivation of the right ventricle in comparison with the left. This question will be discussed more fully in a later section of this article. The exaggerated T and the diphasic character of bundle branch block complexes are equal in importance to the increased QRS interval in their recognition.

Notching of QRS.—We cannot agree with Carter that notching of QRS is uncommon in normal electrocardiograms; we have observed it often and sometimes there is a very complicated multiple notching in Lead III. There is, however, a considerable difference between the notching of normal electrocardiograms and the notching of bundle branch block complexes. In the former, the notching is practically confined to the lead of smallest amplitude (usually Lead III) or at least it occurs relatively close to the base line. In the latter the notch is usually present near the apex of QRS in leads of large amplitude; it may be but often is not most marked in the lead of least amplitude. Although there may not be any essential difference between these two

Fig. 5.—Right bundle branch block produced in dog by cutting right branch of His bundle (Lead II). A, effect of rhythmic stimulation of left apex by break induction shocks. Signal above and also on electrocardiograms. Refractory phase ends between .1650 and .1790 second after Q. B, effect of stimulating right conus region in same way. Refractory phase ends between .2000 and .2104 second after Q.

types of notching, except that of degree, yet we believe that they differ entirely in their practical significance. In order to bring out the reasons for this belief it is necessary to discuss the manner in which notches are produced.

According to Einthoven's [13] scheme of the equilateral triangle for representing the relationship between the three leads (Fig. 6), the height of a deflection in a given lead at a given instant is dependent on two factors; the manifest potential difference (E) which is a definite fraction of the absolute potential difference developed by the heart at that instant, and the cosine of the angle between the direction

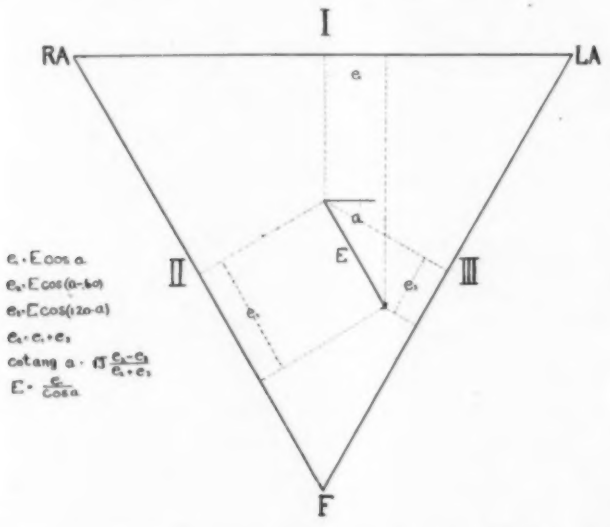

Fig. 6.—The equilateral triangle of Einthoven. I, Lead I; II, Lead II; III, Lead III. R A, right arm; L A, left arm; F, left or right foot. E, manifest potential difference produced by heart at any instant. e1, resulting deflection in Lead I; e2, resulting deflection in Lead II; e3, resulting deflection in Lead III. *a*, angle made by E with I; *a*-60, angle made by E with II; 120-*a*, angle made by E with III.

in which this potential is developed and the line of lead. It may be shown that if the angle made with the line of Lead I is *a*, then the angle made with the line of Lead II is *a*-60 and the angle made with the line of Lead III is 120-*a* (Fig. 6).

13. Einthoven, Fahr, and de Waart: Ueber die Richtung und die manifeste Grösse der Potentialschwankungen in menschlichen Herzen und über den Einflusz der Herzlage auf die Form des Elektrokardiograms, Arch. f. d. ges Physiol. **150**:275, 1913.

During the QRS interval both the scalar magnitude of E and its direction (i. e., angle *a*) are constantly changing. In Figure 7 (the necessary data for the construction of this figure were taken from the

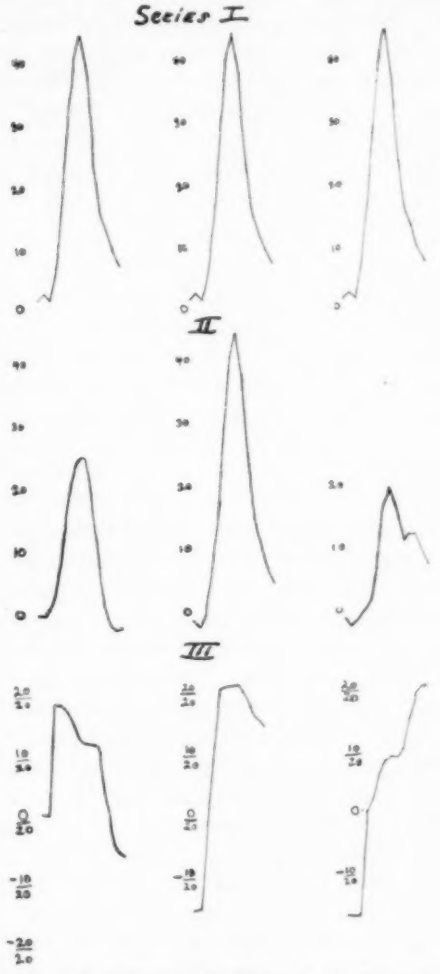

Fig. 7.—(Data taken from paper by Lewis[9].) Series I, three identical curves representing the changes in E (Fig. 6) during QRS interval. Series II, QRS group in Leads I, II and III. Series III, three curves representing changes in cosine *a*, cosine (*a*-60), and cosine (120-*a*) during QRS interval.

work of Lewis[9]) the rapid increase and subsequent decline in the value of E during the QRS interval are shown by the first series of

curves (the three curves are identical). The first curve of the lowest series shows the corresponding variations in the value of cosine a, and the last two curves of this series show the variations in the values of cosine (a-60) and cosine (120-a), respectively. The middle series of curves represents the QRS groups of the three leads in order; the ordinates of these curves are equal to the products of the ordinates of the first series and the corresponding ordinates of the last series.

It is obvious that the changes in a, a-60 and 120-a produced by changes in the direction of the vector E, if measured in degrees, will be the same. The corresponding changes in the cosines of these angles, however, will differ greatly, for the change in cosine per degree is much more rapid near 90 degrees than near zero degrees. Since, moreover, the deflection in a given lead is smallest when the electrical axis is perpendicular to the line of lead (the deflection in Lead I, for example, is smallest when a is 90 degrees) and largest when the electrical axis is parallel to the line of lead, it follows that the QRS group of largest amplitude will most faithfully reproduce the variations in the scalar magnitude of E that take place during the QRS interval; and the QRS of least amplitude will most faithfully record the changes in the direction of the vector E which occur during this interval. Thus QRS of Lead II in Figure 7 closely resembles the curve of series I, while QRS of Lead III resembles it least and shows a distinct notch produced by the irregular movements of the electrical axis.

It will be seen that notching will tend to be produced by irregularities in the movements of the electrical axis under the following conditions: (1) When there is a rapid irregular rotation of the electrical axis toward the perpendicular to the line of lead while the scalar magnitude of E is increasing; (2) when there is a rapid irregular rotation of the electrical axis away from the perpendicular to the line of lead while the scalar magnitude of E is decreasing; (3) when there is a change in the direction of the rotation of the electrical axis from clockwise to counterclockwise or vice versa. Notching in a given lead will occur only when the irregularity in the movement of the electrical axis takes place while the electrical axis is relatively close to the perpendicular to the line of that lead; it will usually be most conspicuous, therefore, in the QRS of least amplitude (Fig. 7). When the heart is activated in the normal fashion, left ventricular effects which tend to cause a counterclockwise rotation of the electrical axis and right ventricular effects which tend to cause a clockwise rotation of the electrical axis occur simultaneously. As the effects of now one and now the other ventricle preponderate, the electrical axis shifts first this way and then that. These frequent and rapid changes in the

position of the electrical axis are mainly responsible for notching of the normal QRS group.

Theoretically, notches may be produced by irregularities in the growth and decline of the manifest potential difference; that is, the curve which represents the variations in the scalar magnitude of E may be notched. Such notches will be reproduced most faithfully in the QRS of greatest amplitude; they may, of course, either be accentuated or diminished by simultaneous irregularities in the movements of the electrical axis which are apt to accompany them. It is probable that the notches which occur on the QRS group of bundle branch block curves are of this type. The curves of right bundle branch block in dogs almost invariably show a distinct notch on the posterior limb of QRS 2 (QRS of Lead II), and it seems probable that this notch is due to a sudden change in the scalar magnitude of E, accompanied perhaps by a sudden change in the direction of the electrical axis, resulting from the sudden spread of the excitation wave in the right ventricle after it has pierced the septum. In fact, we are inclined to believe that this notch is due to the superimposition of Q′ of the dextrogram on the ascending limb of the levogram. A similar but more prominent notch which often occurs near the apex of QRS in left bundle branch block in dogs may be due to the superimposition of R′ of the levogram on the descending limb of the dextrogram.

That the sudden involvement by the excitatory process of a new area of muscle may produce notching is shown by Figure 8. It will be noted that not all the extrasystolic complexes of this figure are of the same form; some of them show a distinct notch on the posterior limb of QRS. All of the extrasystoles shown occurred late in diastole, but it is those which occur latest which show the notch. The position of this notch with reference to the previous P indicates that it occurs after the normal P-R interval, and it is undoubtedly due to the sudden involvement of an area of muscle that is activated by the supraventricular stimulus before the extrasystolic stimulus has reached it.

This curve is an interesting one from another standpoint. It indicates that all of the ventricular muscle has not passed into the refractory state until QRS is complete. The notched extrasystolic complexes show that the muscle which normally first receives the supraventricular stimulus is still irritable when the extrasystolic QRS is almost complete, and the shortened initial deflections and modified final deflections of cycles A 5 and B 1 indicate that the extrasystolic stimulus still finds the surrounding muscle irritable when the normal stimulus has activated a large portion of the ventricular muscle. When the normal stimulus reaches the ventricles after the extrasystolic QRS is complete, however, it finds the ventricles refractory.

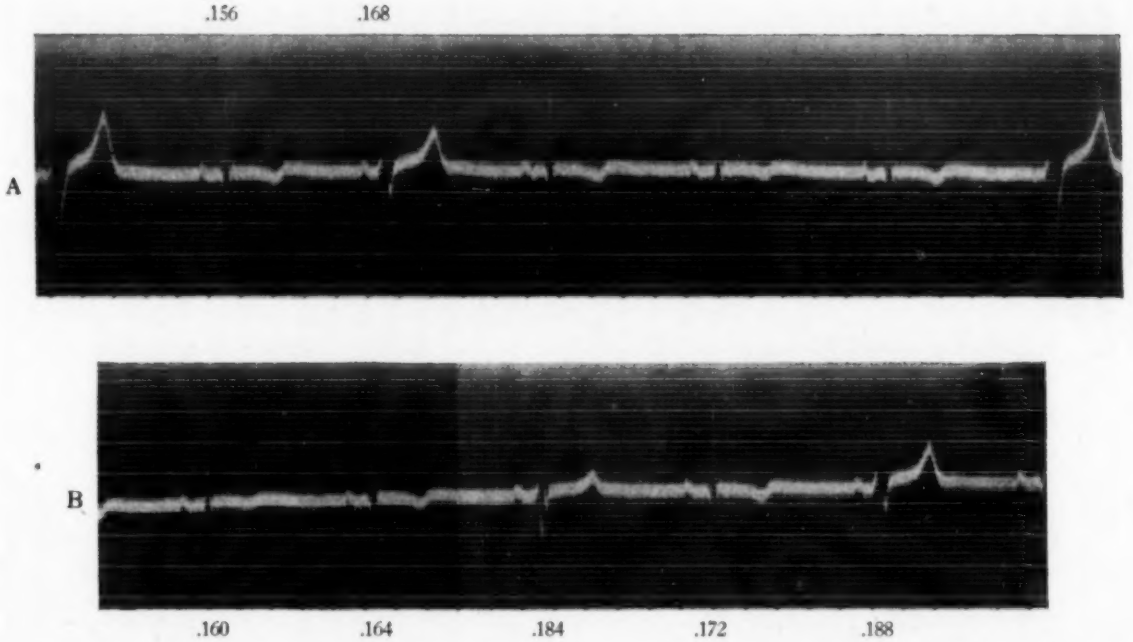

Fig. 8.—Electrocardiogram from apparently normal individual (Lead III). Extrasystoles occurring late in diastole and showing interference between normal and extrasystolic stimuli. Every second complex represents a response partly to the normal and partly to the extrasystolic stimulus.

In concluding the subject of notching, it may be said that it is not an essential feature of bundle branch block complexes; it is frequently absent in man; but when it occurs it is suggestive, especially if it falls near the apex of QRS in a lead of large amplitude.

DIFFICULTIES ENCOUNTERED IN THE DIAGNOSIS OF BUNDLE BRANCH BLOCK

Typical examples of right bundle branch block are recognized without difficulty (Fig. 9). The diphasic character of the ventricular

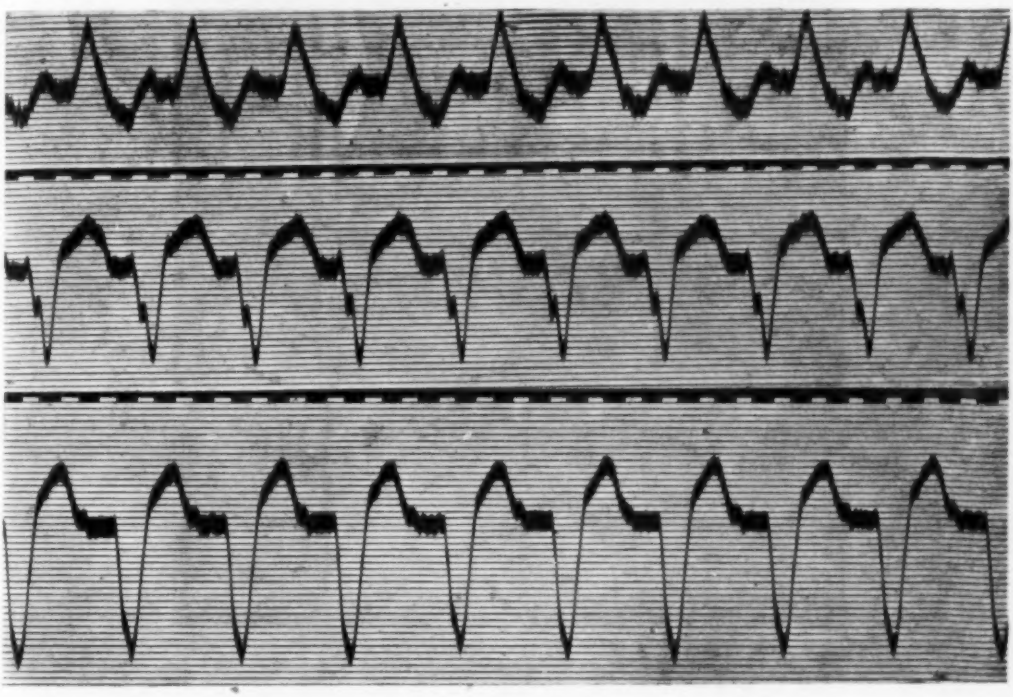

Fig. 9.—Right bundle branch block. QRS equals 0.179 second.

complex, its relatively large amplitude, the notch which so frequently occurs near the apex of QRS, the long QRS interval often measuring more than 0.15 second, are sufficiently distinctive. Comparatively few of the aberrant electrocardiograms observed clinically, however, are typical examples of this condition. Especially confusing are the transitions which are seen between the type of ventricular complex characteristic of left ventricular preponderance (Fig. 10) and the type

characteristic of right bundle branch block (Figs. 9 and 14). Such transitional complexes are shown in Figures 11, 12 and 13. According to Lewis,[9] the direction of the terminal deflection T serves to distinguish between curves of these two types; bundle branch block complexes are typically diphasic, preponderance complexes are not, but show the same variations of T that occur in the normal ventricular complex. One who attempts to interpret large numbers of clinical curves soon finds, however, that at least one-half of the left ventricular

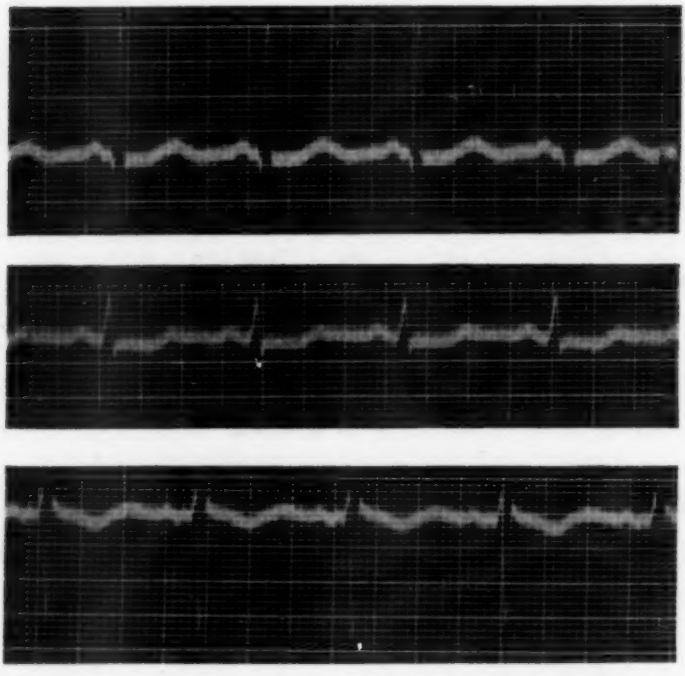

Fig. 10.—Left ventricular preponderance. P-R 0.12, QRS 0.073. T1 positive and T3 negative.

preponderance curves that he encounters show the same type of T as do curves of right bundle branch block; T 1 is negative and T 3 positive, so that the ventricular complex is diphasic in Leads I and III. QRS of Lead II is less frequently negative in preponderance curves than in right bundle branch block, but this difference is not sufficiently distinctive to be of use in differentiating the two types. The QRS interval of preponderance curves is often increased and the transitional curves spoken of show QRS intervals varying from normal to

0.15 second. No sharp line can be drawn, therefore, between the curves of left ventricular preponderance and those of right bundle branch block, and the interpretation of many aberrant electrocardiograms consequently remains doubtful.

The cause of the frequent occurrence of a downwardly directed T 1 and a large upwardly directed T 3 in left ventricular preponderance curves is not known. Perhaps this type of T is a less sensitive indicator of left ventricular preponderance than the well-known deformity of

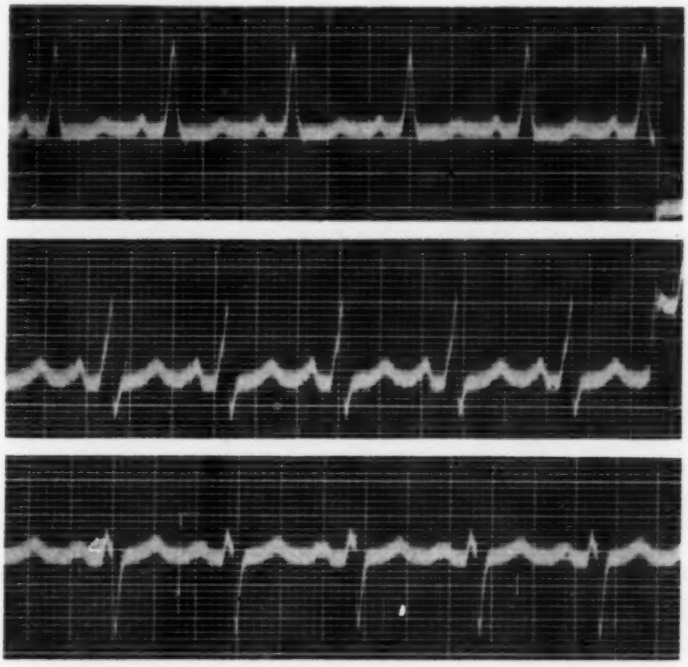

Fig. 11.—Left ventricular preponderance or perhaps slightly defective conduction through right branch of His bundle. P-R, 0.16; QRS, 0.108.

the QRS group which is used as the main criterion of this condition. Nevertheless, many patients with undoubted left ventricular preponderance show T waves of normal type. Inversion of T in Lead I has long been considered a sign of myocardial changes, and Eppinger and Rothberger [2] suggest that it may indicate slightly defective conduction through the right branch of the His bundle. There is some evidence in favor of this view, but far less than enough to make it acceptable. In one instance we have obtained curves of left ventricular preponder-

ance and subsequent curves of right bundle branch from the same individual. T 1 of the preponderance curves was negative, and this might be considered the first sign of the bundle branch block which appeared a few days later; but T 2 was negative in the first and positive in the second set of records, so that this explanation is very doubtful in this instance. Many of the preponderance curves showing the

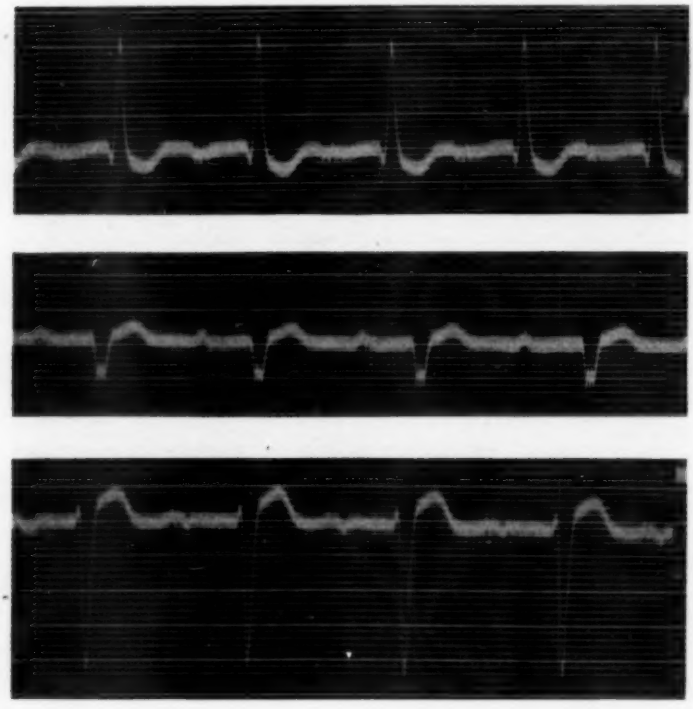

Fig. 12.—Transitional in form between left ventricular preponderance and right bundle branch block. Probably delayed conduction through right branch. P-R, 226; QRS, 0.096.

type of T under discussion have a relatively normal QRS interval which practically rules out disturbances of intraventricular conduction; in others the QRS interval is definitely increased, and when this is the case the explanation offered by Eppinger and Rothberger deserves consideration.

THE EFFECT OF DELAYED CONDUCTION THROUGH THE BRANCHES OF THE HIS BUNDLE ON THE ELECTROCARDIOGRAM

The question of the effect of lesions of the branches of the His bundle of such a character as to delay without completely interrupting the passage of the impulse through them has received less consideration than it merits. When a clamp is placed on the main stem of the His

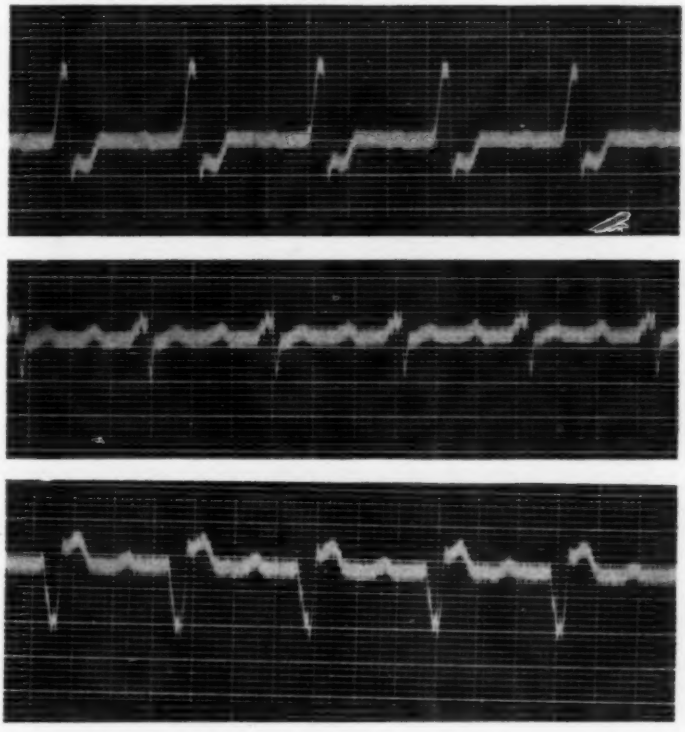

Fig. 13.—Similar to Figure 12, but of smaller amplitude. P-R, 0.24; QRS, 0.114.

bundle and tightened, complete A-V block is produced. If the clamp is then loosened, recovery may take place suddenly or all the stages of partial A-V block may be obtained from frequently blocked auricular beats to simple prolongation of the As-Vs interval. By analogy we might expect similar results from clamping and then releasing the chief branches of the His bundle which have the same structure as the main stem. But the relations are somewhat different. If we produce bundle branch block in this way, the impulse is not prevented, as in

complete A-V block, from reaching the farther side of the blocked area. It does so by passing down the unclamped branch and thence through the ventricular septum to the Purkinje network of the ventricle normally supplied by the clamped branch.

Perhaps for this reason, perhaps for some unknown reason, nothing comparable to the dropping out of ventricular beats in partial A-V block ordinarily occurs as a result of lesions of the branches of the His bundle, so long as the heart is beating regularly. When the heart

Fig. 14.—Complete right bundle branch block. Variations in form of complexes due to forced respiration with changes in position of heart. P-R, 0.14; QRS, 0.142.

is beating irregularly, however, as in auricular fibrillation, variations in the length of the diastolic rest period and consequently in the functional recovery of parts through which the passage of the impulse is delayed but not completely interrupted must occur. When a path is completely destroyed, variations in its rest period cannot affect its conductivity. We may say therefore, that we have complete bundle branch block or delayed conduction through one of the branches of the His bundle,

but we rarely have partial bundle branch block if "partial" be used in the same sense as in A-V block. It may also be stated that the ventricular complexes which indicate delayed conduction through the branches of the His bundle will tend to show variations in form with variations in the length of diastole.

The subject of delayed conduction through the branches of the His bundle has not previously been studied experimentally, and we wish to report briefly here some experiments relating to this subject, reserving the details for a future communication. We produce right bundle branch block in dogs by cutting the right branch of the His bundle. The right ventricle is then stimulated by single induction shocks. When the stimulus falls during the refractory period of the point stimulated no response occurs. When the stimulus falls between T and the following P, a typical right ventricular extrasystole is obtained. When the stimulus falls during the latter part of the P-R interval or on the first part of QRS, the complexes obtained are transitional in form between the extrasystolic complexes and the right bundle branch block complexes. This result might be expected. In right bundle branch block the left ventricle receives the impulse at the normal time, but the activation of the right ventricle is delayed. There is a short period, therefore, during which the left ventricle is passing into the active state while the right ventricle is still quiescent and not yet refractory to stimulation. Stimulation of the right ventricle during this period produces a response, and the resulting ventricular complex is a combination of the normal levogram and the extrasystolic dextrogram. The extrasystolic dextrogram is not usually of exactly the same form as the normal dextrogram obtained by producing left branch bundle block, but it resembles the latter closely in many instances. When the stimulus falls at the proper instant, normal levogram and extrasystolic dextrogram begin simultaneously, and a complex of the normal type results. When the dextrogram precedes the levogram, the same effect is produced as would be produced by delayed conduction through the left branch of the His bundle. When the levogram precedes the dextrogram the same effect is produced as would be produced by delayed conduction through the right branch of the His bundle. We may, therefore, obtain all the transitions between right bundle branch block complexes and the complexes of right ventricular extrasystoles which closely resemble the complexes of left bundle branch block.

We may illustrate our method by some curves from a sample experiment. In this instance an attempt to cut the left branch of the His bundle produced complexes characteristic of left bundle branch block (Fig. 15 B) which persisted for a short period and then gave

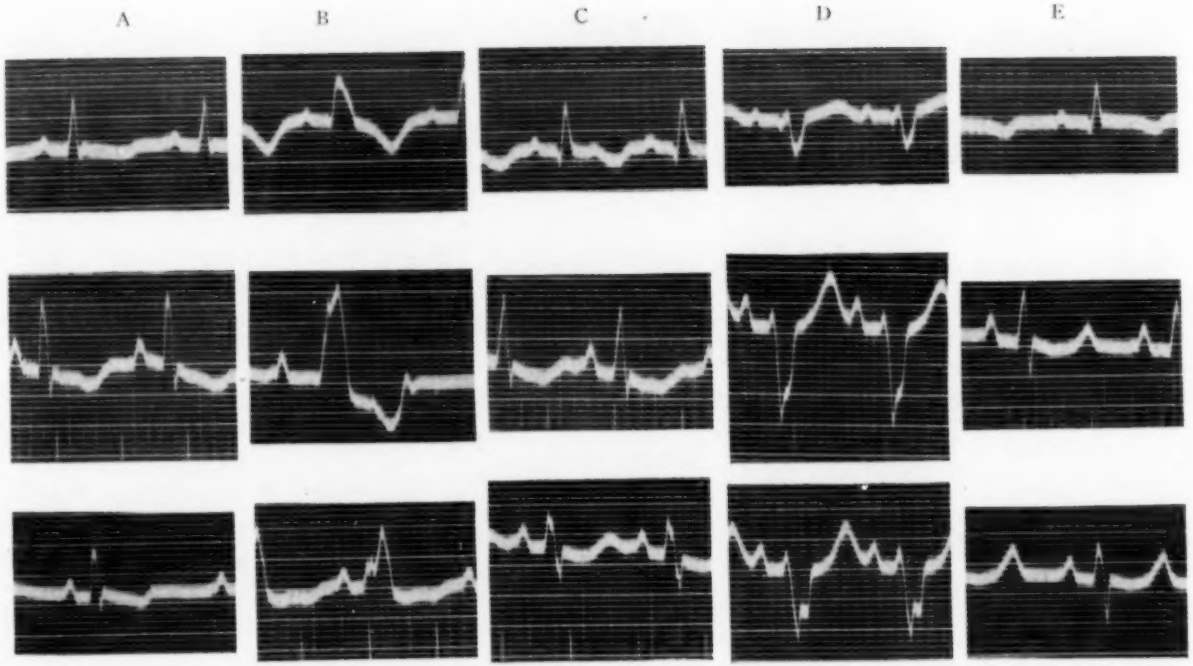

Fig. 15.—A, control curves from dog; B, left bundle branch block; C, recovery; D, right bundle branch block; E, recovery.

place to complexes (15 C) almost exactly like those of the control curves (15 A). Right bundle branch block was then produced by pressure on the right branch of the His bundle; the characteristic complexes (15 D) of this lesion appeared and persisted for about one

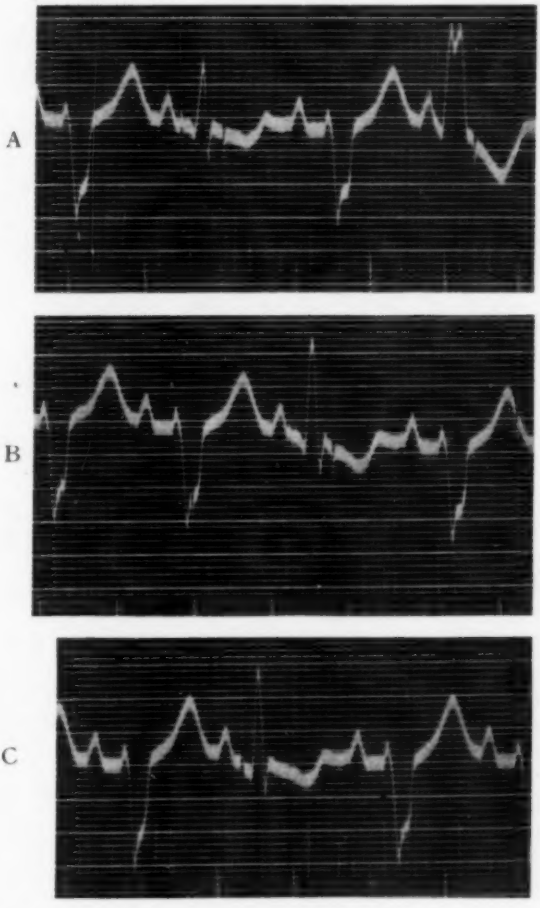

Fig. 16.—Dog 7. Lead II. Right bundle branch block. Reproduction of normal complex by stimuli which fall during P-R interval.

and one-half hours finally giving place to complexes of normal type (15 E). During the time that right bundle branch block was present the right ventricle was stimulated at irregular intervals by single induction shocks. The extrasystolic dextrogram produced (Fig. 16 A)

closely resembled the normal dextrogram obtained earlier in the experiment when left bundle branch block was present. When the stimulus fell at exactly the proper instant the normal ventricular complex was reproduced (Fig. 16 A, B, C). Many complexes transitional in form between the complexes of normal type and those characteristic of right and left bundle branch block were also obtained (Fig. 17). These transitional complexes show the following characteristics: (1) a QRS interval intermediate between that of the normal complexes and that of the bundle branch block complexes, and (2) a T deflection transitional in type between that of the normal complexes and that of the bundle branch block complexes. All the transitional complexes except those which most closely approach the normal complex in form are diphasic and the general resemblance of these complexes to those so-called preponderance curves which are diphasic in Leads I and III, and which show an increased QRS interval, is striking. We are inclined to believe that many of the curves of this type obtained from patients are due to delayed conduction through the right branch of the His bundle.

The transitional complexes obtained by experiments such as the one described above are of value in analyzing the T deflection. We have previously given evidence indicating that the upstroke of T (Lead II) of right bundle branch block curves from the dog is mainly a left ventricular effect due to the early deactivation of the left ventricle in comparison with the right. If it is true, as indicated by our previously mentioned experiments on the duration of the refractory state at various points on the surface of the ventricles in bundle branch block, that the ventricle first activated first becomes inactive, and if it is true that T is due to the deactivation of the ventricular muscle; the upstroke being mainly a left and the downstroke mainly a right ventricular effect (Lead II in dogs); then the direction of T of the transitional complexes under discussion should be mainly dependent on whether the right or the left ventricle is first activated. That this is the case can be verified by an inspection of Figure 17. It will be noted that when the levogram precedes the dextrogram by an appreciable interval, T is positive, and when the dextrogram precedes the levogram by an appreciable interval, A is negative. When left and right ventricular effects are nearly simultaneous the direction of T seems to depend on which of the two ventricles produces effects of greatest amplitude. Furthermore, since the left ventricle is activated by the stimulus which descends from the auricles, the levogram follows the P wave by an interval equal to the P-R interval. The upstroke of T, if it is a left ventricular effect, should also follow P by a definite interval. This is approximately true for all transitional complexes, except those which approach the normal type of complex closely; in

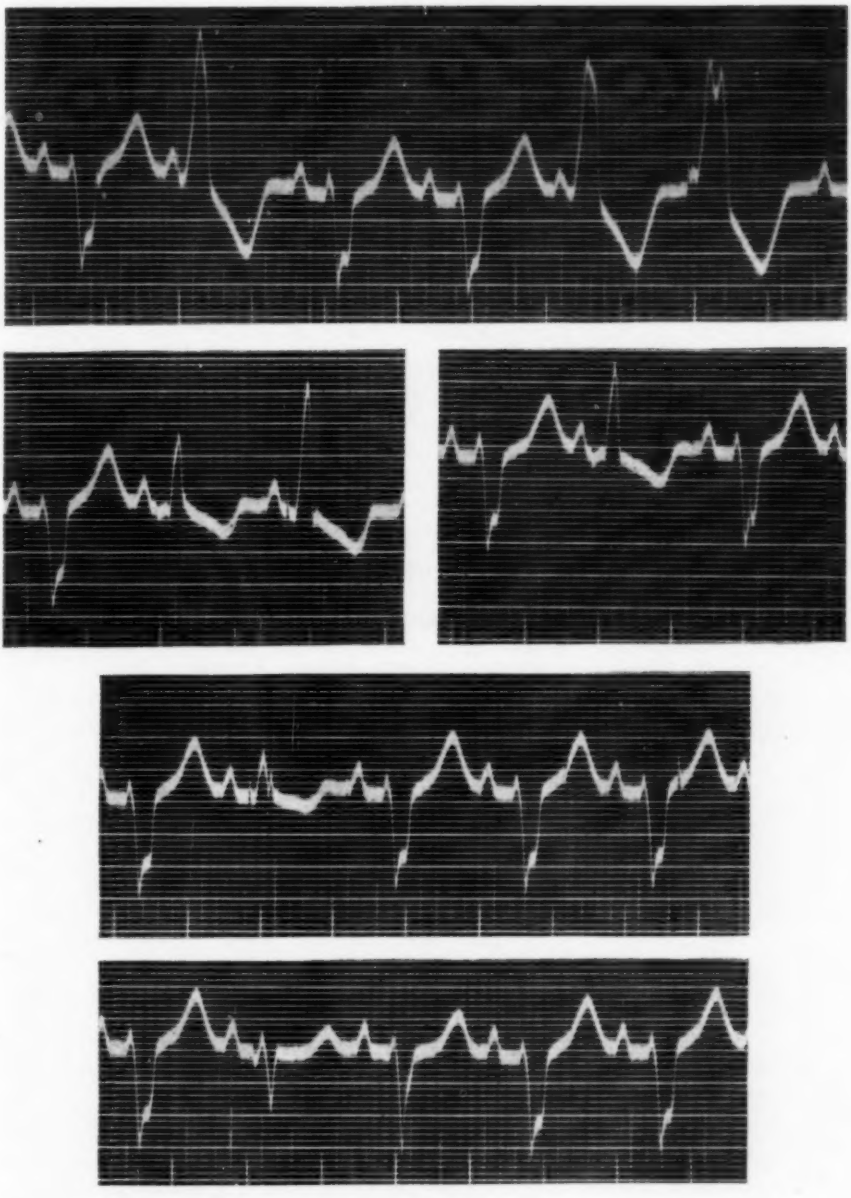

Fig. 17.—Dog 7. Lead II. Right bundle branch block. Production of complexes transitional in form between right bundle branch block complexes and complexes of right ventricular extrasystoles.

these the levogram and dextrogram begin almost at the same time and the corresponding upstroke and downstroke of T are also nearly simultaneous. In such cases the beginning of the upstroke cannot be identified. All the evidence so far obtained indicates, therefore, that the interpretation of T previously given is, in the main, correct.

CLINICAL OBSERVATIONS ON DELAYED CONDUCTION THROUGH THE BRANCHES OF THE HIS BUNDLE

In addition to the experimental evidence given above, we have made certain clinical observations which suggest that diphasic ventricular complexes are often due to delayed conduction through the branches of the His bundle. Figure 18 shows an electrocardiogram from a patient, aged 59, who showed on physical examination cardiac enlargement and arteriosclerosis and who was believed to be suffering from myocarditis. There is a tall R 1 and a deep S 3; T 1 is small and T 3 strongly positive. Lead II resembles Lead III, but is of smaller amplitude. The QRS interval is about 0.1 second. Numerous extrasystoles, auricular and ventricular, are shown. In Figure 19 another curve (Lead III) from the same individual is shown. In this figure there is an interpolated ventricular extrasystole (cycle A_2) of which the QRS interval (0.074) is considerably less than that of the sequential beats (0.1). There is also an auricular extrasystole (cycle A_4) of which the QRS interval (0.116) is considerably greater than that of the sequential beats. In analyzing this electrocardiogram we have used a general principle which may be stated as follows: No single stimulus, in whatever part of the heart it originates, can activate all of the ventricular muscle in a sufficiently short time to give a ventricular complex the QRS interval of which is less than that of the sequential beats unless there is defective conduction through one of the main branches of the His bundle. This statement is based on the following considerations: The QRS interval is a measure of the time taken by the impulse to complete its course over the ventricular muscle. This time will be shortest when the stimulus passes into the conducting systems of both ventricles simultaneously. A stimulus can only reach the conducting systems of both ventricles simultaneously when it arises above the bifurcation of the His bundle or when it arises in the central region of the interventricular septum. Under normal conditions a stimulus which arises in the central part of the septum can hardly pass over the ventricular muscle more quickly than the normal supraventricular stimulus. When there is defective conduction through one of the chief branches of the His bundle, the entrance of the normal stimulus to the corresponding ventricle is delayed so that it can not reach the endocardial surface of both ventricles simultaneously.

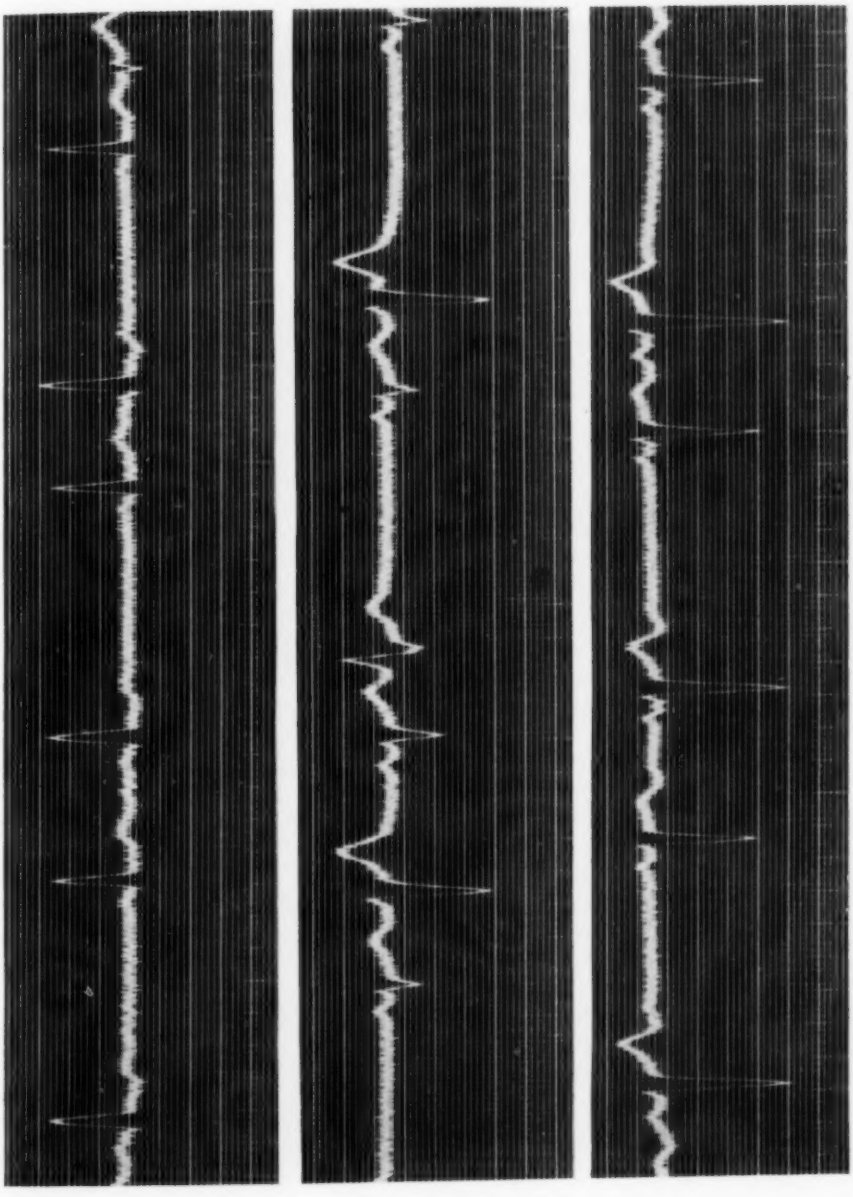

Fig. 18.—E. K. G. 3658. Probably defective conduction through right branch of His bundle. Ventricular extrasystoles. Auricular extrasystoles.

stimulus arising in the septum, however, may still do so, for it may reach the Purkinje system of the affected ventricle below the blocked area.

Assuming that the ventricular extrasystole (cycle A_2, Fig. 19) was due to a single stimulus and not to two stimuli, one arising in each ventricle, which occurred simultaneously, then delayed conduction through the right branch of the His bundle was probably present in the heart from which this curve was obtained. We interpret Figure 19 in the following way. There was defective conduction through the right branch of the His bundle. The stimuli which came down from the auricles had to pass the blocked area to reach the right ventricle; hence the abnormal form of the ventricular complex and its increased QRS interval. The stimulus which gave rise to the auricular extrasystole (cycle A_4) also had to pass the blocked area which had had less time to recover than in the case of the sequential beats; the resulting ventricular complex is more abnormal than that of the sequential beats. The ventricular axtrasystole (A_2) was of septal origin and reached both ventricles at about the same time, but it reached the right ventricle below the blocked area, and the form of the resulting ventricular complex is more nearly normal than that of the sequential ventricular complexes.

In a case published by one of us in 1915,[14] transitions were obtained between the normal complexes and complexes characteristic of right bundle branch block. These transitional complexes must have been due to delayed conduction through the right branch of the His bundle (Fig. 20). From the analysis of a large number of transitions (obtained in Lead III) it was found that changes in the QRS group and in T usually appeared simultaneously. The most constant feature of the transitional complexes was their diphasic character, which was usually evident even in those nearest the normal complexes. The transitional complexes differed from the fully developed bundle branch block complexes principally in their lesser amplitude. As one proceeds from the first abnormal complexes of Figure 20 toward those typical of bundle branch block the amplitudes of QRS and T gradually increase and as these two deflections are in opposite directions the diphasic character of the complexes is gradually accentuated.

The clinical evidence, therefore, is in accord with the experimental evidence that diphasic ventricular complexes are often produced by delayed conduction through the branches of the His bundle.

As to the question of recognizing bundle branch block: It has been pointed out that only a small percentage of the aberrant ventricular

14. Wilson, F. N.: A Case in Which the Vagus Influenced the Form of the Ventricular Complex of the Electrocardiogram, Arch. Int. Med. **16**:108 (July) 1915.

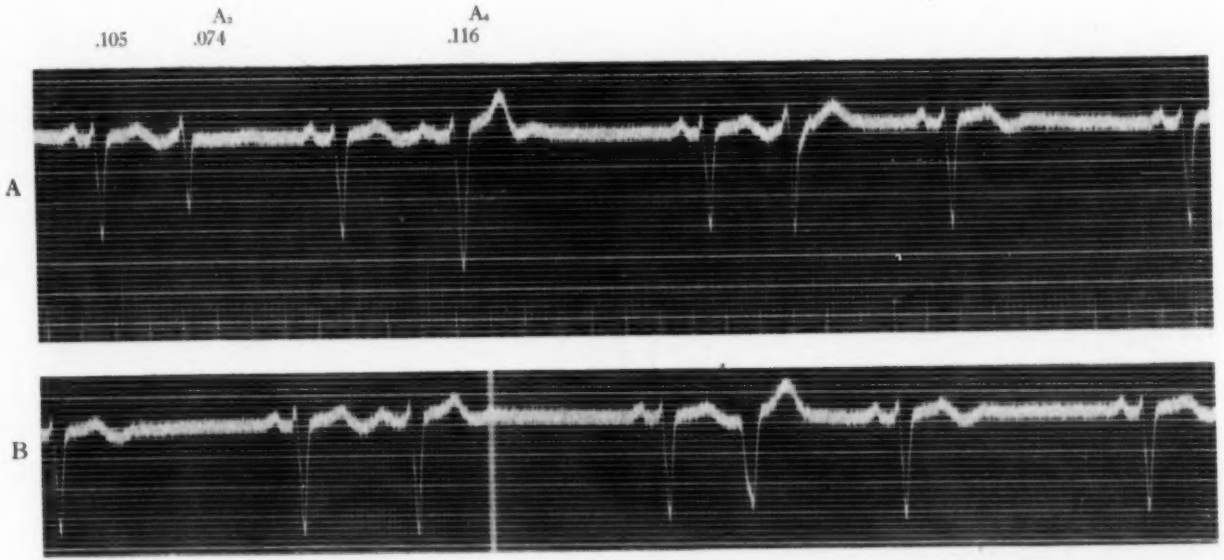

Fig. 19.—Same patient as Figure 18. Lead III. QRS interval of sequential beats 0.105. QRS of interpolated ventricular extrasystole (cycle A_2) 0.074. QRS of auricular extrasystole (cycle A_1) 0.116.

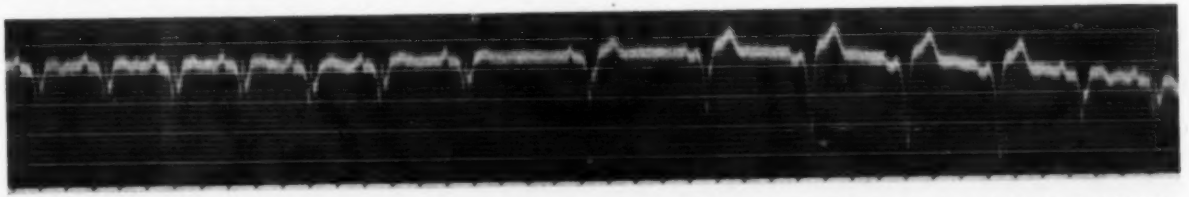

Fig. 20.—Transition from complexes of normal type to complexes characteristic of right bundle branch block (Lead III). Transitional complexes diphasic and of smaller amplitude than bundle branch block complexes.

complexes observed clinically are typical examples of this condition. What are the others? It seems probable that some of them, especially those which show a very long QRS interval, are atypical examples of bundle branch block. There is a great deal of variation in the normal complexes of different individuals due to many causes; variations in the position of the heart; variations in the distribution of the ventricular muscle, and variations in the architecture of the Purkinje system. All of these causes tend also to bring about variations in the ventricular complex of bundle branch block. In addition we are dealing in the case of clinical bundle branch block with pathologic hearts which show much greater variations in the distribution of muscle mass and in position than the hearts of normal individuals. Great variations in the form of the ventricular complex of bundle branch block in man are, therefore, to be expected.

In addition to complete bundle branch block we may have incomplete bundle branch block or what is more properly termed delayed conduction through one of the main branches of the His bundle. We have given clinical and experimental evidence indicating that this may explain many of those aberrant ventricular complexes which are diphasic and which show an increased QRS interval. It is important to note that aberrant complexes of this type are more often of the form to be expected from delayed right than of the form to be expected from delayed left branch conduction; a fact which fits in well with the greater frequency of complete bundle branch block on the right than on the left side.

ARBORIZATION BLOCK

Oppenheimer and Rothschild [15] recently reported a series of cases in which aberrant electrocardiograms were obtained and in which the pathologic findings indicated that lesions of the smaller subdivisions of the His bundle and their arborizations were present. They believe that the abnormality of the ventricular complexes in these cases was due to defective conduction through the finer branches of the ventricular conducting system, and used the term arborization block to indicate this disturbance. The following criteria were given for the recognition of this condition.

1. Abnormal prolongation of the time interval of the QRS group beyond the normal limit of 0.1 second. This prolongation is most manifest in a widening of the R wave so that its foot points are abnormally separated. The R wave no longer has its slender tall spike-like appearance, but is broader and sometimes blunter than normally.

15. Oppenheimer, B. S., and Rothschild, M. A.: Electrocardiographic Changes Associated with Myocardial Involvement, J. A. M. A. **69**:429 (Aug. 11) 1917.

2. Notching of the R wave. This notching may appear on the ascending limb, on both limbs or at the peak. It may be multiple, and its degree and location may vary slightly from beat to beat. In arrhythmias, the shorter the preceding interventricular interval, the more pronounced is the evidence of disturbed intraventricular conduction.

3. Low voltage as expressed by a low amplitude of the waves in all three leads. This change is not uniformly present but when it occurs it helps to differentiate this type from the electrocardiograms of typical bundle branch block.

4. Absence of typical diphasic curves with huge T waves found in experimental bundle branch block.

The pathologic findings need not be described in detail; they included coronary arteriosclerosis with occlusion of the anterior descending branch of the left coronary artery and patchy sclerosis involving mainly the subendocardial muscle and Purkinje tissue. The changes were grossly more marked in the left ventricle than in the right.

EXPERIMENTAL LESIONS OF THE SMALLER SUBDIVISIONS OF THE HIS BUNDLE AND THEIR EFFECT ON THE FORM OF THE ELECTROCARDIOGRAM

By the term arborization block, Oppenheimer and Rothschild refer to defective conduction beyond the subdivision of the two chief branches of the His bundle. The experimental evidence bearing on this problem is of interest. In attempts to cut the left branch of the His bundle which spreads out fanwise over the left surface of the septum many experiments are failures, for it is rarely possible to cut the left branch before it subdivides. In many instances the cut transects only a portion of the fan. Eppinger and Rothberger publish one curve from such an experiment. It is definitely abnormal and of unusual form; on vagus stimulation typical left bundle branch block resulted. It was found that the cut had transected the posterior limb of the fan and had slightly injured the anterior limb. Hemorrhage into the uncut portion of the bundle was apparently responsible for the complete bundle branch block which ensued coincidentally with vagus stimulation. This experiment does not necessarily indicate that section of the posterior limb of the left branch produces profound changes in the form of the ventricular complex, for the anterior limb was also injured and the curves obtained may be interpreted as being due to delayed conduction through the left branch as a whole. Lewis does not mention the effect of cutting the subdivisions of the left branch; but in the paper written with Rothschild,[10] the statement is made that section of that portion of the right branch which bridges the ventricular cavity in the dog produces a diminution of R and an increase of S in the axial electrocardiogram. We have cut minor subdivisions of the left branch in many, and major subdivisions of

the left branch in a few instances, and we have in one instance cut all those subdivisions of the right branch which bridge the ventricular cavity, and we have never observed more than slight changes in the form of the electrocardiogram as a result of these lesions. Such changes as did occur did not resemble, in any way, the changes believed to result from so-called arborization block. There is, therefore, no evidence yet available to show that experimental lesions of the larger subdivisions of the branches of the His bundle produce more than minor changes in the form of the electrocardiogram. Nor would striking changes be expected for the subdivisions of the branches of the His bundle anastomose very freely,[10] and it is difficult to see how any lesion of these structures, except an extremely extensive one, could greatly delay the passage of the excitation wave over the ventricular muscle or could greatly change its course.

The effect of diffuse lesions involving large areas of the Purkinje network is a different matter. The only experiments bearing on this problem are those published by Lewis and Rothschild.[10] They showed that a cut completely transecting the endocardial surface of the conus region may delay the activation of the conus muscle by as much as 0.02 second. The delay is comparatively slight; the region chosen is a particularly favorable one for the purpose, for the cut can be so made as to transect all or nearly all of the Purkinje plexus supplying the conus muscle. The experiments indicate that the excitation wave travels through the Purkinje system more rapidly than through the ordinary ventricular muscle. They do not necessarily indicate that patchy lesions of the subendocardial tissues, such as Oppenheimer and Rothschild describe, may produce striking changes in the form of the ventricular complex.

It should be noted that Smith,[16] who did a large number of experiments in which he ligated various branches of the coronary arteries, obtained extensive lesions of the subendocardial tissues, but did not observe abnormalities of the electrocardiogram similar to those attributed to such lesions by Oppenheimer and Rothschild.

In conclusion, it must be admitted that there is little experimental evidence to indicate that lesions of the subdivisions of the branches of the His bundle or their arborizations produce striking or characteristic changes in the form of the electrocardiogram. Until such evidence is brought forward the diagnosis of arborization block rests on a very insecure foundation.

16. Smith, F. M.: The Ligation of the Coronary Arteries with Electrocardiographic Study, Arch. Int. Med. **22**:8, 1918.

SUMMARY

Complete bundle branch block produces characteristic changes in the form of the ventricular complex both in animals and in man.

Delayed conduction of the impulse through the branches of the His bundle (incomplete bundle branch block) produces ventricular complexes which are transitional in form between the normal ventricular complex and complexes characteristic of complete bundle branch block.

The T deflection is produced by the deactivation of the ventricular muscle. In Lead II of the dog the upstroke of T in right bundle branch block is mainly a left ventricular effect; the downstroke of T in left bundle branch block is mainly a right ventricular effect.

There is little experimental evidence to indicate that lesions of the subdivisions of the branches of the His bundle or their arborizations produce those changes in the form of the ventricular complex usually attributed to arborization block. Until such evidence is brought forward the diagnosis of arborization block rests on an insecure foundation.

A CASE EXHIBITING SLOW AURICULOVENTRICULAR RHYTHM AND PAROXYSMAL TACHYCARDIA WITH UNUSUAL ABILITY TO INTERRUPT THE FAST RATE*

M. H. FUSSELL, M.D., AND C. C. WOLFERTH, M.D.
PHILADELPHIA

INTRODUCTION

Tracings from clinical cases exhibiting auriculoventricular rhythm are comparatively rare, although the reports of Belski,[1] Cowan, Fleming and Kennedy,[2] and Hume[3] suggest that the condition is not uncommon, at least as a transient manifestation, in certain more or less acute diseases that especially attack the heart, such as diphtheria, rheumatic fever and subacute endocarditis. It is undoubtedly rare in chronic cardiac disease, and is usually, but apparently not always, associated with extensive myocardial involvement. It may also be produced experimentally in normal persons by various disturbances of the inhibitory apparatus.

Englemann[4] was the first to recognize auriculoventricular rhythm, following his application of the first Stannius ligature in the frog. Lohmann[5] obtained the rhythm in the mammalian heart. Since that time, a large amount of experimental work has been done, culminating in the studies of Meek and Eyster,[6] who proved the actual auriculoventricular nodal origin of the rhythm by the method of initial negativity. These investigators, working on dogs, concluded that the inherent automaticity of the auriculoventricular node, although subject to wide individual variations, averaged about two-thirds that of the sinus node; when impulses did not arrive from above at a more rapid rate than they were being formed at the auriculoventricular node, the latter tended to assume the cardiac autonomy.

Experimentally produced auriculoventricular rhythm may be classified into two types, according to the method by production. Type I, which has been most studied, is produced by depression or abolition

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* Cardiographic studies from the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania.
* Read before the Annual Meeting of the Association of American Physicians, May 4, 1920.

1. Belski: Ztschr. f. klin. Med. **67**:67, 1909.
2. Cowan, Fleming and Kennedy: Lancet **1**:227, 1912.
3. Hume: Heart **5**:25, 1913.
4. Engelmann: Arch. f. Anat. u. Physiol., Phys. Abth., p. 505, 1903.
5. Lohmann: Ibid., p. 431, 1904.
6. Meek and Eyster: Heart **5**:227, 1914; Arch. Int. Med. **18**:775 (Dec.) 1916.

of automaticity at the sinus node, or interference with conduction of impulses to the auriculoventricular node. Consequently, it is a relatively slow rhythm. Type II, brought on by exaltation of automaticity of the auriculoventricular node, was first produced by Rothberger and Winterberg [7] by stimulation of the left accelerator nerve. It is, therefore, relatively more rapid than sinus rhythm.

The above mentioned types of experimentally produced auriculoventricular rhythm, according to Lewis' [8] classification, are of the ectopic hemogenetic type. Most of the clinical cases are also of this type. The majority are due to depressive influences exerted above the auriculoventricular node, although some are doubtless aided in their production by the effect of accelerator influences on the node. As far as we are aware, no clinical case of the homogenetic type has been demonstrated to be due solely to acceleration of auriculoventricular stimulus production, although the work of Rothberger and Winterberg would seem to show that such a case would be theoretically possible.

The paroxysmal junctional tachycardias, although ectopic heterogenetic rhythms and consequently of fundamentally different character, are also classified as auriculoventricular rhythms. This group is rare, eight probable cases being reported. The characteristics of paroxysmal tachycardia distinguish them from homogenetic rhythms, but it has been impossible to differentiate absolutely some of the cases of functional origin from paroxysmal auricular tachycardia with delayed conduction, as in Lewis' [9] second case. Lewis' [10] first case and Cohn's [11] case, however, appear to be clear-cut and establish the condition as an entity.

The case we wish to report shows the following features of interest:

1. The length of time shifting of pacemaker back and forth between the sinus and auriculoventricular nodes was observed.
2. The variations in rate during auriculoventricular rhythm.
3. Alterations in As-Vs relations.
4. Attacks of paroxysmal tachycardia with unusual ability on the part of the patient to interrupt the fast rate.
5. The unusual behavior of the heart following the interruption of the tachycardia.
6. The resemblance of the tracings of the tachycardia, in a case which had previously shown auriculoventricular rhythm, to those of paroxysmal junctional tachycardia.

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7. Rothberger and Winterberg: Arch. f. d. ges. Physiol. **135**:559, 1910.
8. Lewis: Quart. J. Med. **6**:221, 1913.
9. Lewis: Heart **2**:127, 1910.
10. Lewis: Heart **1**:306, 1910.
11. Cohn: Heart **2**:170, 1910.

REPORT OF CASE

Mrs. F. R., aged 62, was seen Dec. 21, 1919, at the Hospital of the University of Pennsylvania.

Present Complaint.—Palpitation of heart. The patient states that she has had attacks of palpitation of the heart since childhood. At that time she would get attacks of rapid heart beating after severe exertion. These attacks lasted a few minutes and disappeared quickly. She nevertheless took part in the ordinary plays of childhood. Gradually these attacks of palpitation increased. For the past two years she has been practically invalided.

The attacks begin often by clutching in the throat which she says last two days. Occasionally this would arouse her out of sleep, and at that time she would have sudden palpitation. This sudden palpitation makes her faint if she attempts to rise. Occasionally she has blackness before her eyes. From the very first of these very severe attacks of cardiac palpitation, the action could be controlled if she would hold her breath for a considerable length of time. When they could not be so controlled, when they ended they would end suddenly just as suddenly as they began. Occasionally, preceding an attack, she would have dimness of vision. Sometimes the attacks would come on after walking, sometimes as has been said, in her sleep. She has had slight swelling of her legs. Vertigo rather constantly, and vertigo always accompanies the severe cardiac distress. She has shortness of breath on the least exertion lately. Within the past six months she has been considerably worse and came to the hospital for rest. She has some aching in the region of her heart but no distinct pain. If an attack of palpitation of the heart came on while she was walking, she could usually reach home from a moderate distance, but with difficulty.

Physical Examination.—The eyes are entirely normal. There is no arcus senilis. There is slight edema of the skin over the body. Nothing abnormal is noticed about the ears. There was no glandular enlargement. She had some capped teeth, but apparently the teeth in general are in good condition. Her neck and throat are normal. The chest is normal in shape. She is spare for a woman of her general size. There is marked pulsation of the vessels of the arms and neck. Expansion of chest is normal. Abdomen is normal in shape.

The cardiac impulse is feeble. The apex beat is best felt just inside of the nipple line. Dulness of the heart is at the right edge of the sternum, at the upper border of the third rib and midclavicular line. There is a loud systolic murmur over the aorta and sternum, and also over the region of the apex. This murmur is conducted into the axilla. During this examination there is no arrhythmia. The first sound is almost entirely replaced by the murmur. Second is not unduly accentuated. The lungs are entirely normal and abdominal examination is entirely normal. At this first examination her heart was beating at about 50 a minute and was perfectly regular. Jan. 6, 1920, she was seen in an attack of tachycardiaa. The cardiac impulse was diffuse, but the dulness was apparently not increased. On auscultation, the heart's action was regular, the murmur which was loud during the slow action of the heart was difficult to hear. She was asked to hold her breath, which she did, and immediately after one or two attempts of interruption of the rhythm, the heart slowed down and continued at the rate of about 50 a minute while the examiner was in the room.

All of the laboratory tests were normal.

Apparently, the case was one of ordinary paroxysmal tachycardia in a heart which acted slowly between the paroxysms. Again, there was the unusual ability for her to control the attack of paroxysmal tachycardia by holding her breath, and the time between the first slow beats seemed unusually long, as though some block were occurring.

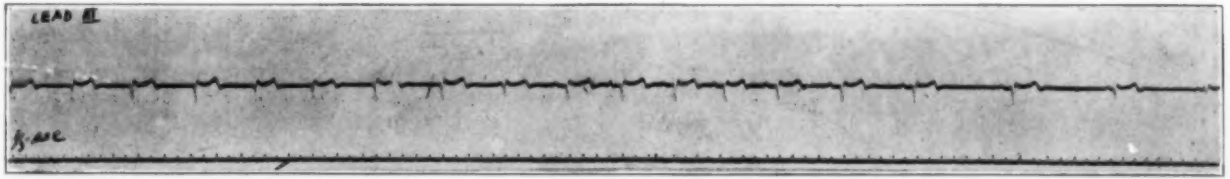

Fig. 1 A.—Lead III. Taken Jan. 24, 1920. First eight cycles. Auriculoventricular rhythm, rate about 60. No P wave visible. Spontaneous short period of sinus rhythm. Only slight conductivity defect; the P-R interval does not exceed 0.20 second. Slow auriculoventricular rhythm following sinus rhythm. Patient had received atropin sulphate, 1/100 grain, eight times in the preceding forty-eight hours and showed physiologic effects.

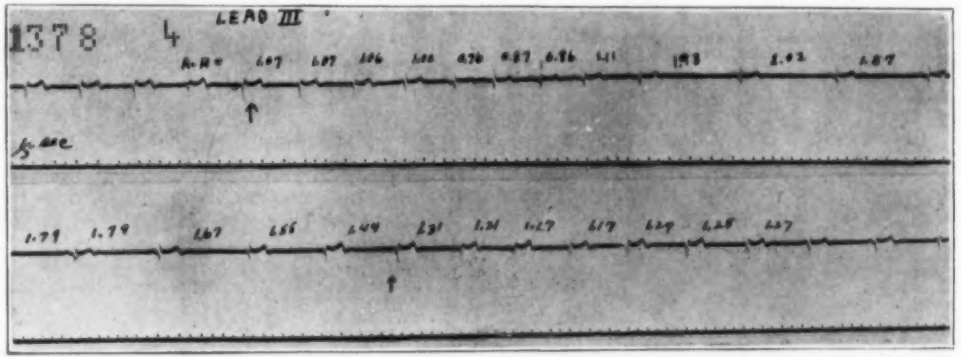

Fig. 1 B.—Lead III. Taken immediately after Fig. 1 A. Deep breath at point indicated by first arrow, held until point indicated by second arrow. Alteration in ventricular complex while breath was held. This tracing shows the typical variations in rate of the auriculoventricular rhythm and their relations to a period of sinus rhythm.

After about two weeks' rest in bed, the heart slowed down and the patient left the hospital January 31, very much more comfortable. She had not had an attack of tachycardia for two weeks.

She went from Philadelphia to Montrose in a comfortable car, developed a severe distressing cough with fever, and in about a week began to manifest signs of cardiac decompensation. She was seen in Montrose with a typical attack of decompensation, the heart beating about 160, dyspnea, large liver, edema of the lungs, and finally death occurred in two weeks after the beginning of this condition. No necropsy was held.

OBSERVATIONS ON THE SLOW AURICULOVENTRICULAR RHYTHM

Tendency for the Rhythm to Alternate Between the Sinus Node and the Auriculoventricular Node.—During the entire period of four weeks, while electrocardiographic studies were being made, a constant shifting of the pacemaker back and forth between the sinus and auriculoventricular nodes was observed, except during the periods of tachycardia. Tracings show from two to twenty cycles of normal rhythm, followed by transition to the slow rhythm, then abrupt change to the normal rhythm again. The periods of auriculoventricular rhythm observed varied from about thirty seconds to at least several minutes. Absolute rest and quiet favored longer periods of slow rhythm. Mild excitement, such as caused by applying electrodes, increased the frequency of periods of normal rhythm. Pressure on the vagi or ocular pressure sometimes cause shifting of the pacemaker back to the sinus node, but the results were inconstant. Once, a slight cough was followed instantly by return of normal rhythm, sometimes deep breathing produced the same result. Under varying amounts of atropin, the tendency for the pacemaker to shift spontaneously was not abolished.[12]

Variations in Rate During Auriculoventricular Rhythm.—The transition to auriculoventricular rhythm was sometimes preceded by a slowing of the sinus rhythm (Figs. 1 A and 1 B), sometimes it came on following an auricular extrasystole (Fig. 2 A), but frequently it occurred abruptly (Fig. 2 B) without preceding slowing of the sinus rhythm or extrasystoles. As a rule, after two or three cycles with increasing R-R intervals, the rate reached from 30 to 45, and with minor variations it tended to increase gradually, until eventually it became fairly steady around from 50 to 60, until the onset of the next period of sinus rhythm.

Owing to the spontaneous variations in rhythm, the case was not favorable for study of changes due to vagal influences. Pressure on the neck, ocular pressure and deep breathing produced no appreciable

12. Atropin sulphate was administered by mouth, 1/100 grain q. i. d., for one day, and t. i. d. the next four days. All statements in respect of atropin effects refer to observations made during this period.

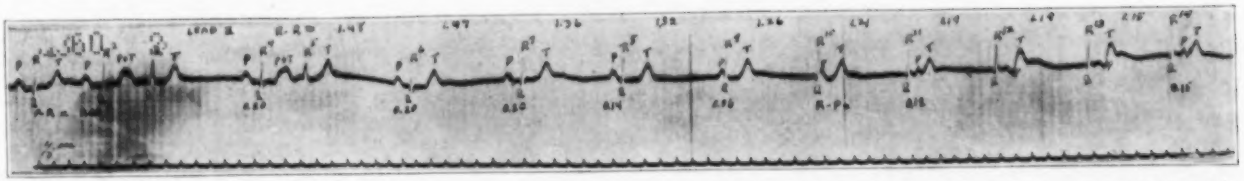

Fig. 2 A.—Lead II. Taken Jan. 2, 1920. First two cycles end a short period of sinus rhythm, followed by extra systole with delayed conduction. The beginning of auriculoventricular rhythm interrupted by another extrasystole. Gradual development of R-P sequence. Gradual acceleration of rate. (The patient had received tincture of digitalis, 10 minims by mouth the evening before and again on the morning this tracing was made.)

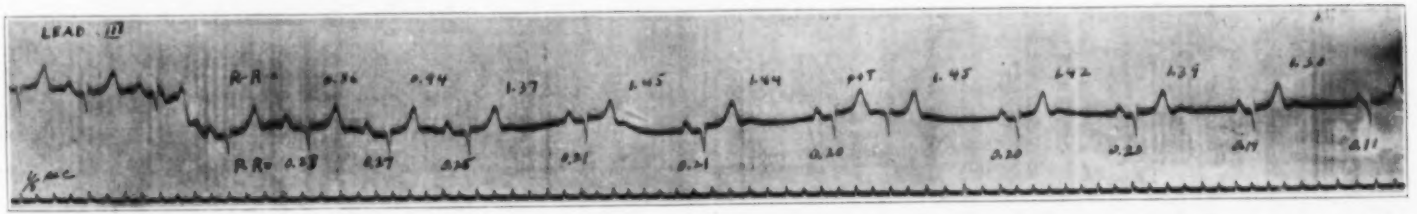

Fig. 2 B.—Lead III. Taken immediately after Figure 2 A. First seven cycles end a period of normal rhythm showing depressed conductivity. Abrupt onset of auriculoventricular rhythm. Gradually decreasing P-R intervals. Tendency toward acceleration of rate during auriculoventricular rhythm.

change except in so far as they sometimes initiated sinus rhythm. The atropin administration also was without effect apparently on the rate of the auriculoventricular rhythm.

The Relations of Auricular Systole to Ventricular Systole. — A slight depression of conductivity, which was not entirely abolished by atropin, was noted in all tracings, and at no time during sinus rhythm was the P-R interval less than 0.20 second. In Figure 2 it varies but reaches a maximum of 0.27 second. In connection with the onset of auriculoventricular rhythm, the P-R interval shows an immediate slight decrease. In general, after maintaining this relation for a few cycles, P gradually approaches R until it becomes lost in the Q R S group, sometimes emerging again to the left for a few cycles (Fig. 1 B, last cycle). The changes in P-R intervals occur usually in relation to changes in rate, the intervals decreasing toward zero as the rate increases. However, this relation is not constantly observed. Figure 2 A shows R-P intervals, and is the only tracing in which they were obtained.

Neither pressure on the vagi, ocular pressure, deep breathing nor the administration of atropin produced any appreciable changes in the relation of P to R.

OBSERVATIONS ON THE TACHYCARDIA AND THE INTERRUPTIONS PRODUCED BY THE PATIENT

The tracing shown in Figure 3 was taken during one of the paroxysms of tachycardia. The rate was about 135, the rhythm regular and the ventricular complexes, with minor differences in Lead II are similar to those occurring during sinus rhythm, except for the presence of the P wave between the Q R S group and the T wave.

For years the patient had been able to inhibit the tachycardia temporarily, and occasionally terminate an attack, by taking a deep breath and holding it. This maneuver was not always successful, but frequently a cycle of events, of which Figure 4 shows an example, occurred. After the breath was held for from ten to fifteen seconds, the tachycardia would terminate suddenly, sometimes with a moderately long pause followed by an isolated contraction, then an extremely long pause of from four to five seconds (Fig. 5 A), while sometimes the longest pause would occur immediately after the interruption of the tachycardia (Fig. 4). Following the long pause, contractions of supraventricular origin, but showing no evidence of P wave, occurred irregularly with single contractions or short runs of sinus origin interspersed. When the period of interruption lasted longer, a fairly regular slow auricular ventricular rhythm became established, inter-

rupted by short runs of sinus rhythm, similar in every way to the behavior of the heart observed apart from the paroxysms of tachycardia.

The position of the P wave with reference to the ventricular complex during offset and onset and during the period of interruption is

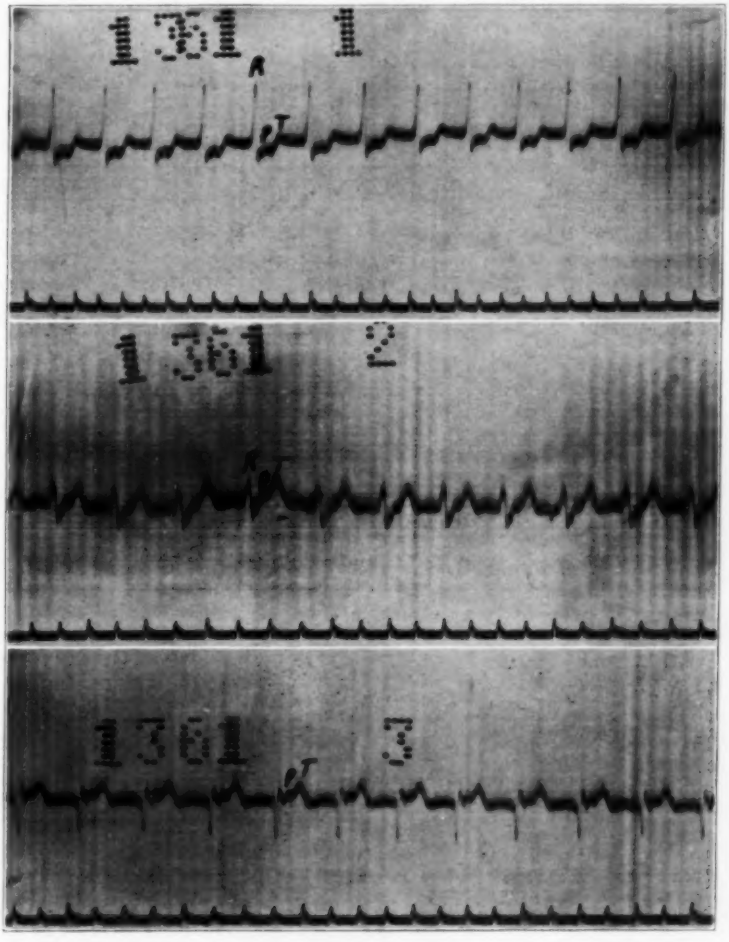

Fig. 3.—Taken Jan. 3, 1920. Shortly after the onset of the tachycardia. P wave between Q R S complex and T wave in all three leads.

of some interest. Figures 5 A and B show no evidence of auricular activity within the last ventricular complex before the offset. In Figure 5 A the first cycle after the offset has a P-R interval of 0.38

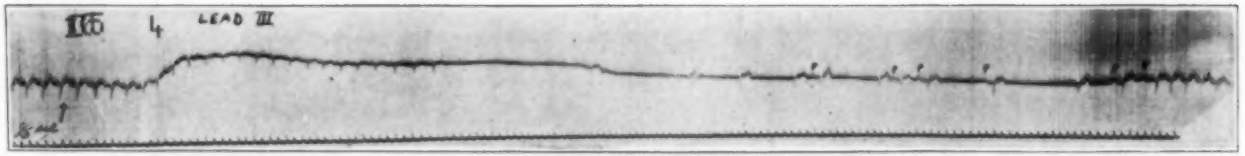

Fig. 4.—Lead III. Taken Jan. 7, 1920. Shows sequence of events following interruption of fast rhythm. Deep breath taken at point indicated by arrow. Held until patient was aware of changed rhythm. Long pause of 4.5 seconds followed by ventricular escape. No regular rhythm established. Long P-R interval at onset of tachycardia.

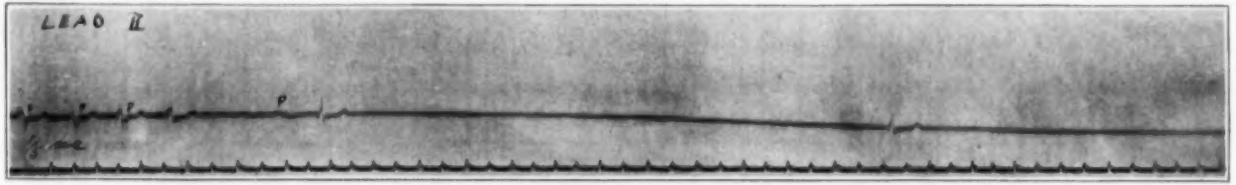

Fig. 5 A.—Lead II. Taken Jan. 5, 1920. Interruption of tachycardia. Last ventricular complex before offset does not show deformity due to P wave. Next contraction shows long P-R interval. The long pause lasts 4.9 seconds.

Fig. 5 B.—Lead III. Taken immediately after Figure 5 A. Interruption of tachycardia. Last ventricular complex does not show deformity due to P wave.

second. In Figure 4, during the interruption, the longest P-R interval observed in cycles of sinus origin is 0.28 second. The tachycardia is initiated again by a cycle with a P-R interval of 0.37 second.

COMMENT

The Slow Rhythm.—While frequent alternations of the pacemaker back and forth between the sinus and auricular nodes has been observed in several cases, usually, as in those reported by Lewis [8] and Laslett,[13] the rates of stimulus production in the two centers are fairly close, so that Laslett's suggestion of variations in vagal tone as the determining factor in the shift, might well apply. In our case, attempts at stimulation of the inhibitory apparatus appeared to have some effect, but not a constant one, in bringing about a few cycles of sinus rhythm, or at least hastening the recurrence of such a period; however, the sinus rhythm would not persist whether the stimulation be continued or not. On the other hand, attempts to remove inhibitory control by atropin did not abolish the shifting back and forth of the pacemaker. The very slow rate following the sinus rhythm revealed the fact that the ability to produce stimuli in the sinus node must have been almost, if not completely, exhausted temporarily, and despite any nervous influences that could be brought to bear, required at least some rest to bring about a restoration of the function. The main effect of nervous influences was apparently to alter the length of the period before the node became able to discharge impulses again.

Changes in relation of auricular systole to ventricular systole during auriculoventricular rhythm have been studied experimentally by several observers. Hering [14] suggested that they were due to migration of the pacemaker. Zahn [15] and Meek and Eyster [6] demonstrated that migration of the pacemaker was accompanied by change in As-Vs intervals. According to the latter, coronary sinus rhythm was accompanied by only slight shortening of As-Vs time, but when the impulse arose in the lower or ventricular portion of the node, the ventricles might contract even before the auricles. Lewis, White and Meakins [16] have shown that reversed block might also account for changes in As-Vs relations during auriculoventricular rhythm, not only to the extent of producing Vs-As intervals, but to failure of transmission of impulse back to the auricles. White [17] was able to increase R-P intervals in his case by vagal stimulation and by digitalis, and to decrease the intervals lengthened by digitalis through

13. Laslett: Heart **6**:81, 1915.
14. Hering: Arch. f. d. ges. Physiol. **136**:446, 1910.
15. Zahn: Ibid. **151**:247, 1913.
16. Lewis, White and Meakins: Heart **5**:289, 1914.
17. White: Arch. Int. Med. **16**:517 (Oct.) 1915.

the administration of atropin. These effects were produced presumably by modifying reversed conduction. In those clinical cases without depression of conductivity, spontaneously changing As-Vs relations are probably dependent largely if not wholly on migration of the pacemaker. Weil [18] ascribed the alterations in As-Vs relations observed in his tracings to this factor.

In our case, the facts suggest that both migration of the pacemaker and depressed conductivity are involved in the changing As-Vs relations, as seen in Figure 2 A and B. Explanation on the basis of independent auricular and ventricular contractions falling near each other is easily excluded, for even if it were granted that both auricles and ventricles might be contracting at the same slow rate, it would be unreasonable to expect them to maintain their relations in view of the irregularities in rhythm that occur.

P-R intervals up to 0.27 second during the sinus rhythm, evidence well marked depression of conductivity. The fact that P-R intervals during the early cycles of auriculoventricular rhythm may still be 0.20-0.21 second (Fig. 2 A and B) demonstrates that the principal defect in conductivity lies beyond the point of formation of impulses, consequently a slight forward block is evident. But even complete recovery of conductivity could not account for the development of the R-P intervals. On the other hand, numerous tracings taken later, when conductivity was improved, as in Figure 2, A and B, show not only shorter P-R intervals at the beginning of the auriculoventricular rhythm but no R-P relations at all. Consequently, the only explanation that seems adequate to account for the facts observed is the assumption that the region of the defect of conductivity is within the auriculoventricular node; at the beginning of auricular rhythm, it lies beyond the pacemaker and slight block is present; but by the migration of the pacemaker to lower levels of the node, it is passed and slight reversed block occurs.

The Tachycardia.—The tracings shown in Figure 3 admit of two possible interpretations: (1) paroxysmal junctional tachycardia with R-P intervals, or (2) paroxysmal auricular tachycardia with delayed conduction. The explanation is furnished by the tracings showing interruption and onset of the rhythm (Figs. 4 and 5, A and B). The last ventricular complex before offset shows no P wave as it would if the rhythm were of junctional origin, while the onset is initiated by a cycle with a P-R interval of 0.37 second, demonstrating that conduction is delayed sufficiently for the auricular systole of a particular cycle to fall within the ventricular systole of the preceding cycle during the tachycardia.

18. Weil: Deutsch. Arch. f. klin. Med. **116**:486, 1914.

The fact that conduction is so much more delayed during the tachycardia than occurred with sinus rhythm, is not to be explained in this case on the basis of depression of conductivity due to more frequent transmission of impulses. The first cycle of tachycardia shows the same marked delay in comparison with the contractions of sinus origin that occur just before it (Fig. 4). The same delayed conduction is seen when auricular extrasystoles interrupt either sinus or auriculoventricular rhythm (Fig. 2, A and B). A possible explanation is furnished by the work of Eyster and Meek,[6] who show that conduction from the sinus to the auriculoventricular node occurs normally not by spreading of the impulse through the mass of auricular tissue, but by specialized conduction paths; while conduction by the auricle may be accomplished, it is difficult and prolonged. Consequently, whether ectopic auricular impulses are carried to the auriculoventricular node through the mass of auricular tissue, or back to the sinus node and thence through the sinoventricular conduction paths the As-Vs time might be prolonged.

The mechanism by which the patient was able to interrupt the tachycardia was not clear. It has long been known that stimulation of the inhibitory apparatus may interrupt a paroxysm of tachycardia, but as shown in the case studied by Cohn and Fraser,[19] this usually occurs promptly. We were unable to initiate the change by pressure on either vagus or by ocular pressure. Carter and Wedd[20] exhibit tracings in which the tachycardia was thought to be interrupted by the aid of some voluntary effort on the part of the subject. In our case the fact that holding the breath for from ten to fifteen seconds at least, was necessary, raises the question as to whether slight asphyxial influences might temporarily depress the irritability of the ectopic focus in a heart with evident advanced myocardial disease and doubtless a precarious blood supply.

The unusual behavior of the heart following the interruption of the tachycardia, which, so far as we are aware, is unique, was probably due, in part at least, to the easy exhaustion of stimulus production in the sinus node, which was pointed out above as the principal factor concerned in the shifting back and forth of the pacemaker during the periods of slow rhythm. Whether still further depression of the sinus node was caused by the same mechanism that depressed the irritable focus in the auricle could not be determined. Evidently, however, the automaticity of the auriculoventricular node was markedly disturbed and required time for its recovery so that a regular auriculoventricular rhythm could be established only during the longer periods of interruption of the tachycardia.

19. Cohn and Fraser: Heart **5**:93, 1913.
20. Carter and Wedd: Arch. Int. Med. **22**:571 (Nov.) 1918.

SUMMARY

A case is reported with a history of attacks of paroxysmal tachycardia occurring over a period of more than forty years; an ability to interrupt the fast rate with temporary cessation of cardiac activity; periods of slow rhythm with interspersed short runs of about the normal rate; abundant clinical evidences of myocardial disease and decompensation.

The electrocardiographic tracings showed left ventricular preponderance, depressed conductivity, periods of sinus rhythm alternating with auriculoventricular rhythm, and paroxysmal auricular tachycardia.

Observations were made in respect of the alternations of the pacemaker between the sinus and auriculoventricular nodes, variations in rate, relations of auricular systole to ventricular systole. The significance of these phenomena is discussed.

The paroxysmal tachycardia was determined as auricular in origin by analysis of curves of offset and onset. The method by which the patient obtained interruption of the tachycardia is described, and an attempt made to evaluate the factors responsible for the behavior of the heart during the period of interruption.

EXPERIMENTAL OBSERVATIONS ON THE ATYPICAL Q-R-S WAVES OF THE ELECTROCARDIOGRAM OF THE DOG *

FRED M. SMITH, M.D.
CHICAGO

In the last few years considerable interest has been shown in the atypical Q-R-S waves of the electrocardiogram. These abnormal electrocardiograms have usually been seen in patients who gave clinical evidence of a failing myocardium. Many have died from cardiac failure within a few months following the time these abnormal electrocardiograms were discovered. In those cases that came to necropsy there has usually been found an extensive fibrosis of the heart which often involved the endocardial and subendocardial layers.

Until recently it was generally believed that these atypical Q-R-S waves were due to a lesion of one or the other of the main branches of the auriculoventricular bundle. This conception was based primarily on the experimental work of Eppinger and Rothberger.[1] They produced experimental lesions of the right and left branch of the auriculoventricular bundle. Those hearts with a lesion of the right branch gave an electrocardiogram in which the ventricular phase resembled that of a premature contraction rising in the base of the left ventricle. Those with lesions of the left branch caused ventricular waves similar to those of a premature contraction originating in the apex of the right ventricle. In the former instance the R-wave in Lead I and the S wave in Lead III are those encountered in preponderance of the left ventricle, while in the latter the S wave in Lead I and R wave in Lead III are those of hyperbalance of the right ventricle. Later Cohn and Lewis,[2] Mathewson [3] and also Carter [4] reported clinical electrocardiograms resembling those produced experimentally by Eppinger and Rothberger, accepting the interpretation of the latter investigators. In some instances, the patients came to necropsy, and histologic examination of the hearts showed lesions of the right branch of the auriculoventricular bundle.

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* From the Medical Department of Rush Medical College and Presbyterian Hospital.

* This investigation was aided by funds given by Mrs. C. H. McCormick and R. T. Crane, Jr.

1. Eppinger and Rothberger: Ztschr. f. klin. Med. **50**:1, 1910.
2. Cohn and Lewis: Heart **4**:15, 1912.
3. Mathewson: Heart **4**:385, 1913.
4. Carter, E. P.: Clinical Observations of Defective Conduction in the Branches of the Auriculoventricular Bundle. A Report of Twenty-Two Cases in Which Aberrant Beats Were Obtained, Arch. Int. Med. **13**:803 (June) 1914.

In 1917, Oppenheimer and Rothschild [5] reported a series of sixty-two cases with atypical Q-R-S waves. A careful histologic study was made in eleven hearts obtained from necropsies. They found a disseminated patchy sclerosis which in most instances involved the endocardial and subendocardial layers of the heart. They concluded that the abnormal electrocardiograms were due to lesions of the branching fibers of the auriculoventricular bundle, and suggested that the condition be known as "arborization block." Recently Carter,[6] Willius,[7] Neuhof [8] and Wedd [9] reported cases, accepting the interpretation suggested by Oppenheimer and Rothschild.

In 1916, Robinson [10] reported cases in which the broad and notched R waves of the electrocardiogram changed to the normal form while the patients were at rest and under the effects of digitalis. He suggested at this time that possibly fatigue of the heart muscle might be a big factor in the production of these abnormal ventricular waves. Later clinical observations,[11] the work of Fletcher and Hopkins [12] on the effect of diminished oxygen supply on muscle activity, and the experiments of Mines [13] and of Burridge [14] on the effect of change in hydrogen ion concentration on conduction through the ventricles, led Robinson to advance the hypothesis that these abnormal electrocardiograms were caused by an accumulation of acid metabolites in the ventricular structures.

The diversity of opinion as to the interpretation of these atypical waves has led to the investigation which forms the basis of this report. In this work the abnormal electrocardiograms under consideration were produced experimentally in the dog.

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5. Oppenheimer, B. S., and Rothschild, M. A.: Electrocardiographic Changes Associated with Myocardial Involvement, J. A. M. A. **69**:429 (Aug. 11) 1917.

6. Carter, E. P.: Further Observations on the Aberrant Electrocardiogram Associated with Sclerosis of the Atrio-Ventricular Bundle Branches and Their Terminal Arborizations, Arch. Int. Med. **22**:331 (Sept.) 1918.

7. Willius, F. A.: Arborization Block, Arch. Int. Med. **23**:431 (March) 1919.

8. Neuhof, S.: The Clinical Significance of the Abnormally Wide Ventricular Deviation in the Electrocardiogram, Arch. Int. Med. **22**:45 (July) 1918.

9. Wedd, A. M.: The Clinical Significance of Slight Notching of the R-Wave of the Electrocardiogram, Arch. Int. Med. **23**:515 (April) 1919.

10. Robinson, G. C.: The Relation of Changes in the Form of the Ventricular Complex of the Electrocardiogram to Function Changes in the Heart. Arch. Int. Med. **18**:830 (Dec.) 1916.

11. Robinson, G. C.: The Significance of Abnormalities in the Form of the Electrocardiogram, Arch. Int. Med. **24**:422 (Sept.) 1919.

12. Fletcher and Hopkins: Lactic Acid in Amphibian Muscle, J. Physiol **35**:247, 1907.

13. Mines: On Dynamic Equilibrium in the Heart, J. Physiol. **46**:349 1913.

14. Burridge: Propagation of Contraction in the Frog's Heart, J. Physiol. **45**:6, 1912.

METHOD

The anesthesia, artificial respiration and the method of exposing the heart were the same as employed in former work.[15] Lesions of the branches of the auriculoventricular bundle were produced by incising the endocardium of the ventricles. A small knife with a long oval shank was adopted for this work. This knife was introduced into the ventricular cavity through the wall at a point nearly opposite that area of the endocardium that we wished to incise. That point of the cardiac wall to be pierced by the knife on introduction into the

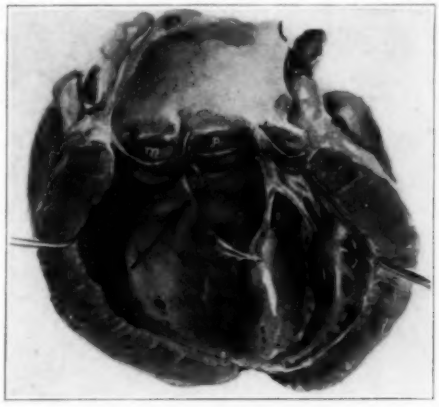

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Fig. 1.—Dog 2. 1. The arrow points to an incision between the middle and posterior cusps of the aortic valves. The left branch of the auriculoventricular bundle was cut. The right branch soon failed to carry auricular impulses. See electrocardiogram, Figure 1. 2. Shows the point in the left ventricular wall through which the knife was introduced into the left ventricular cavity. This figure also shows the branches of the left coronary arteries that were ligated to hasten the dilatation of the left ventricle.

ventricular chambers was first enclosed by a purse-string suture. This suture was closed immediately following the withdrawal of the knife. Very little blood was lost from the ventricular chambers by this precaution. Electrocardiograms were taken prior to and at varying intervals following the incision of the endocardium.

15. Smith, F. M.: The Ligation of Coronary Arteries with Electrocardiographic Study, Arch. Int. Med. **22**:8 (May) 1918.

I. CASES IN WHICH BOTH BRANCHES OF THE AURICULOVENTRICULAR BUNDLE WERE CUT

In four instances the interventricular septum was slit transversely just below the aortic valves. In two dogs the ventricles immediately assumed a much slower rhythm independent of that of the auricles. This slow rhythm did not develop in the third dog until a period of ten minutes had elapsed. In the fourth dog the ventricles immediately came to a standstill in diastole. They were stimulated to a slow rhythmical contraction by massage. In all instances the ventricles seemed to be laboring under great difficulty following the onset of the slow rhythm. The contraction wave, however, seemed normal. Within one hour the hearts of these four dogs became markedly dilated and cyanotic. The contractions of the ventricles at this time were distinctly modified. In some areas no contraction could be detected. Finally, both ventricles came to a standstill, except for a small area in the region of the interventricular septum. This condition was usually followed in a few minutes by ventricular fibrillation.

At necropsy, a transverse slit was found through the septum just below the aortic valves in three instances. Both branches of the auriculoventricular bundle had been cut. In the fourth dog a slit was found in the interventricular septum between the middle and posterior cusps of the aortic valves (Fig. 1). The left branch of the auriculoventricular bundle was cut and the right soon ceased to function, as shown by complete heart block that developed.

The electrocardiograms taken immediately following the incision of the septum showed some change in the Q-R-S group. The R wave was slightly broad at the base, and in one case notched. In one instance there was a broad and notched S wave. The T wave assumed the direction opposite to that of the R and S waves. The electrocardiogram became more abnormal as the ventricular contraction became more modified and the degree of heart block more pronounced (Fig. 2 [3]). The most atypical electrocardiograms were obtained at that time when the contraction of the ventricles was confined to the vicinity of the interventricular septum (Fig. 2 [4 and 5]).

II. CASES IN WHICH INCISIONS WERE MADE IN THE ENDOCARDIUM OF THE LEFT VENTRICLE

In eleven instances, the endocardium of the left ventricle was incised. The contraction of this chamber was apparently not affected until dilatation was produced by ligation of the branches of the left coronary artery. In three cases the left ventricle dilated without our having ligated coronary arteries. These were instances in which the incisions were extensive and involved the deeper layers of the muscu-

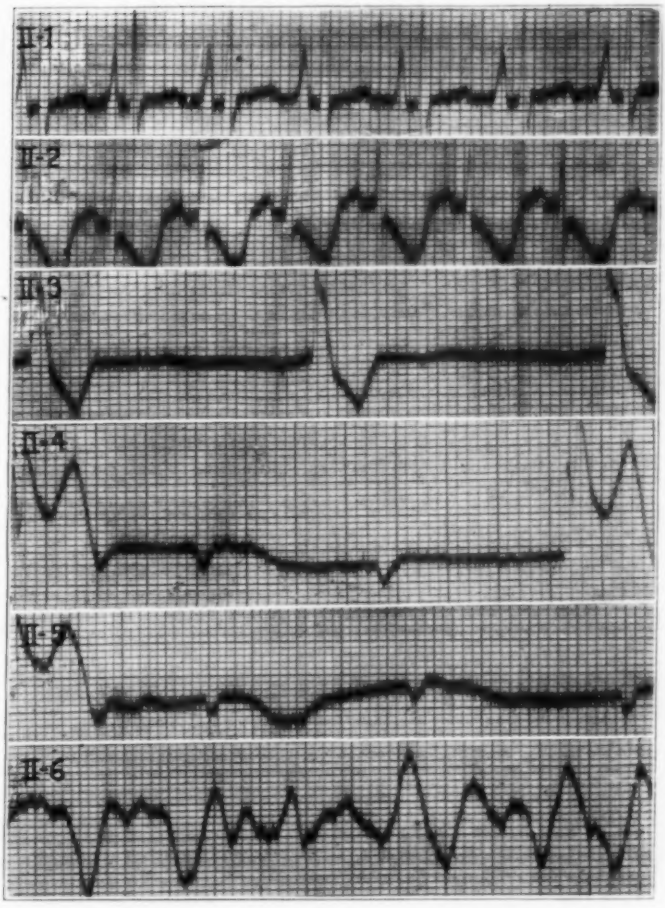

Fig. 2.—Dog 2. Lead II. 1. Initial electrocardiogram. 2. Record taken immediately after a slit was made in the interventricular septum between the posterior and median cusps of the aortic valves as shown in Figure 1. The T-wave is now a negative phase. 3. This electrocardiogram was taken after the onset of the slow rhythm—complete heart block. Note that the R-wave is notched and broad at the base. The heart had begun to dilate. 4. This electrocardiogram is even more abnormal. The ventricles were now markedly dilated and the contraction very atypical. 5. This record was taken as the condition of the heart was growing progressively worse. 6. Ventricular fibrillation.

Fig. 3.—Dog 10. Arrows point to transverse incisions in the endocardium just below the aortic valves. See electrocardiogram, Figure 16.

Fig. 4.—Dog 13. The arrows point to incision parallel and median to the posterior papillary muscle and also transverse incisions below the aortic valves. The largest of the latter incisions divided the left branch of the auriculo-ventricular bundle. See electrocardiograms, Figures 6 to 10.

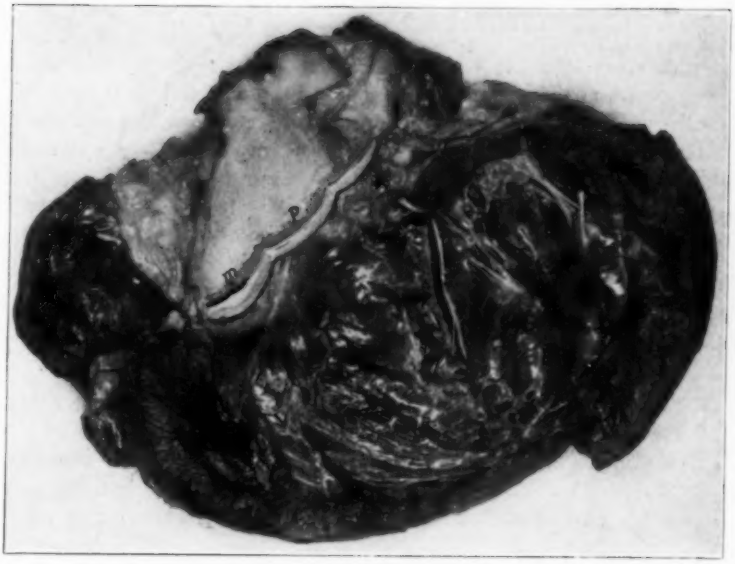

Fig. 5.—Dog 11. An incision in the endocardial and subendocardial layers parallel and median to the posterior papillary muscle. See electrocardiogram, Figure 11.

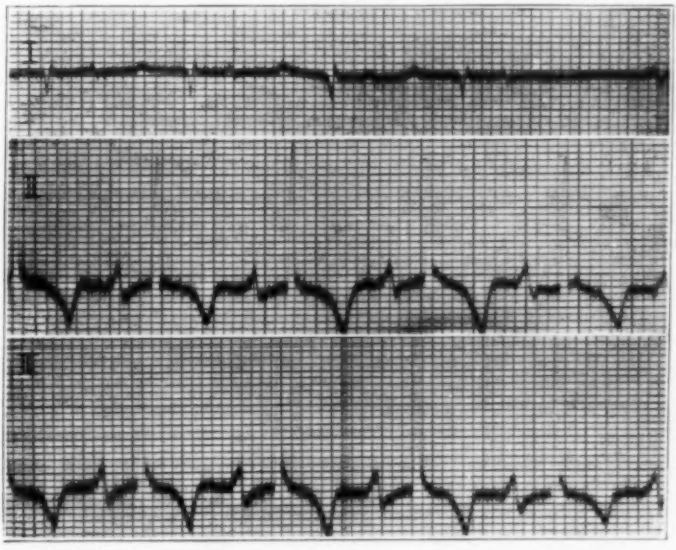

Fig. 6.—Dog 13. Initial electrocardiogram. The P-R interval is slightly prolonged.

lature. When the left ventricle became dilated and cyanotic, areas began to come to a standstill especially in the lateral and posterior basal regions. Later the contraction of both ventricles became limited to the vicinity of the interventricular septum as in those hearts in which both branches of the auriculoventricular bundle were cut.

At necropsy the incisions were found to vary as to extent and location. They were all located between the aortic valves and the posterior papillary muscle. In most instances there were two or more small transverse incisions just below the aortic valves (Figs. 3 and 4). Figure 4 shows in addition a long incision parallel to the posterior papillary muscle. In three cases there were incisions median and parallel to the posterior papillary muscle (Fig. 5).

Electrocardiograms taken immediately after extensive incisions had been made in the endocardium showed very little change in the Q-R-S group (Figs. 6, 7, 8 and 11 [2]). After the left ventricle became dilated and the contraction modified, the R wave was broad at the base and often notched (Figs. 9 and 11 [3]). Later this wave became more atypical (Fig. 11 [4 and 5]). In no instance was there an S wave in Lead I.

III. A CASE IN WHICH THE RIGHT BRANCH OF THE AURICULOVENTRICULAR BUNDLE WAS CUT

In one instance the right branch of the auriculoventricular bundle was cut. The contraction of the right ventricle immediately became less than before the incision. In about twenty minutes this chamber stopped contracting except for a small area in the region of the interventricular septum. Later both ventricles became dilated and began fibrillating.

At necropsy, a longitudinal slit was found in the interventricular septum which extended up to the aortic valves (Fig. 12). Near the upper pole of this incision there was a communication between the ventricles that had been made by the knife. Although histologic examination was not made, it was impossible to see how the right branch of the auriculoventricular bundle escaped being cut.

The electrocardiogram taken immediately following the incision showed a prominent S wave in lead III (Fig. 14). This wave was slightly broad at the base, but not notched. This type of electrocardiogram is generally regarded as indicative of left ventricular preponderance. In this instance the preponderance of the left ventricle was due to a lessening of the contraction of the right ventricle. The electrocardiogram became more abnormal (Fig. 15) as the contraction of the right ventricle diminished and was most atypical just prior to the onset of ventricular fibrillation.

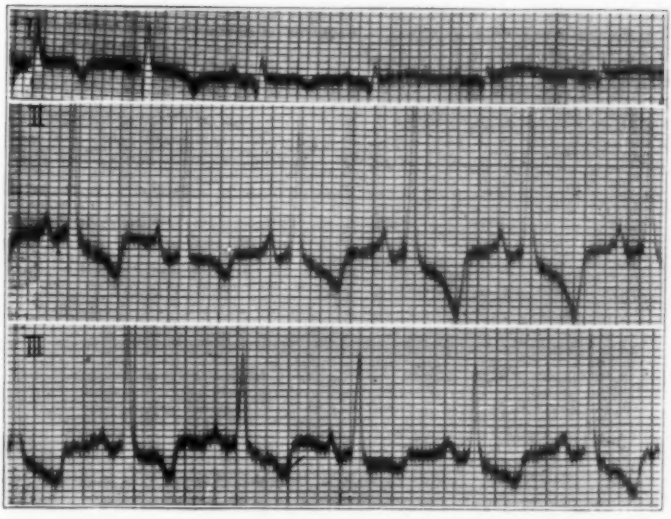

Fig. 7.—Dog 13. This record was taken immediately after an incision was made parallel and medial to the posterior papillary muscle as shown in Figure 4.

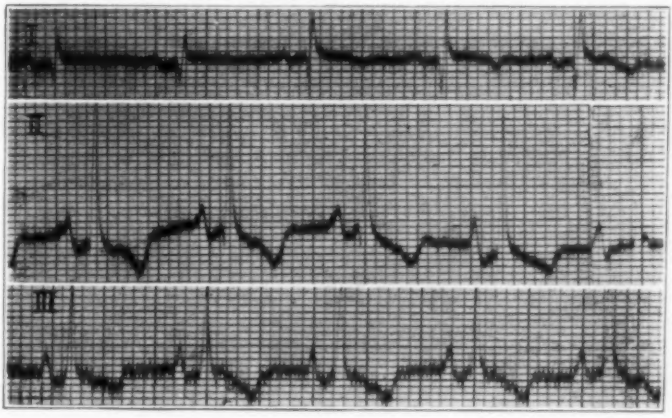

Figure 8.—Dog 13. This electrocardiogram was taken after that shown in Figure 7. There are no changes in the Q-R-S waves even though the conduction paths to the greater part of the left ventricles have been divided.

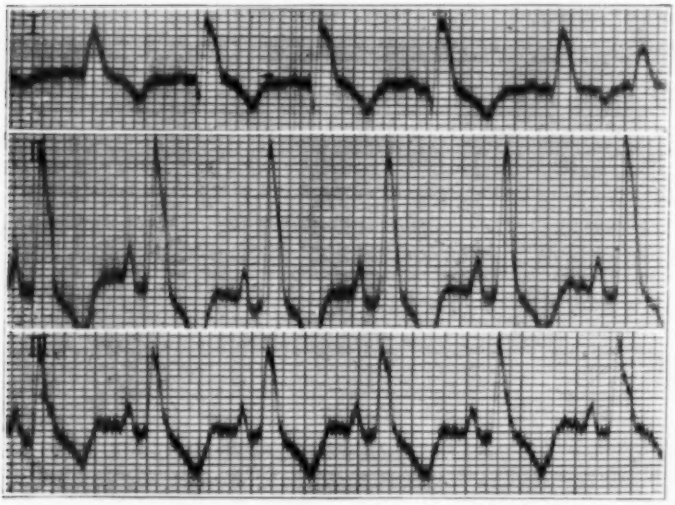

Fig. 9.—Dog 13. Electrocardiogram taken after the transverse incisions had been made in the endocardium below the aortic valves as shown in Figure 4. The heart was dilated at this time and there were areas in the posterior and lateral basal regions of the left ventricle in which there were no apparent contractions.

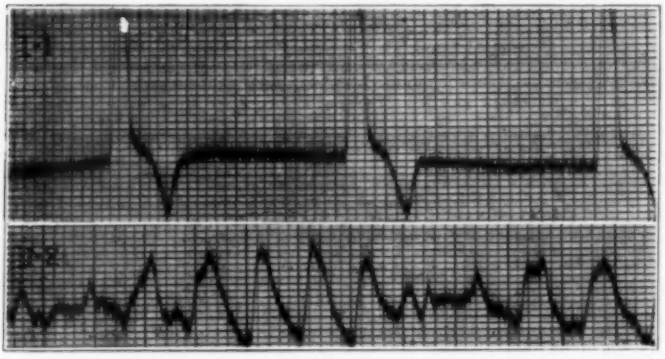

Fig. 10.—1. Dog 13. Lead II. This record was taken ten minutes following that shown in Figure 9. The auricles are now fibrillating. 2. This electrocardiogram was taken five minutes after that shown in 1—ventricular fibrillation.

COMMENT

There are reasons for believing that the "arborization block theory" advanced by Oppenheimer and Rothschild falls short of explaining the abnormal Q-R-S waves. In a former investigation [15] extensive fibrosis of the endocardial and subendodocardial layers of the heart of the dog was produced by the ligation of the coronary arteries. Many of these dogs were allowed to live for a period of two or three

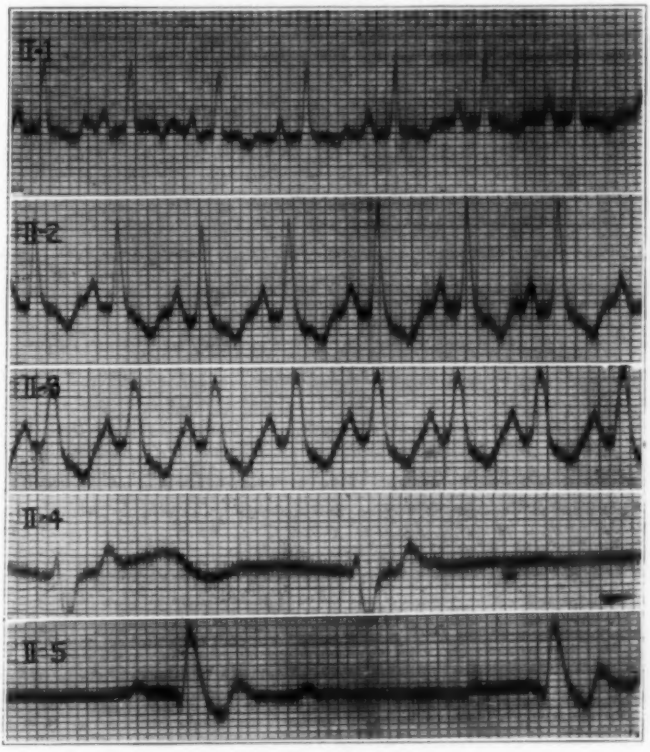

Fig. 11.—Dog 11. Lead II. 1. Initial electrocardiogram. 2. This record was taken twenty minutes after an incision was made in the endocardial and subendocardial layers median to and parallel to the posterior papillary muscle as shown by Figure 5. The R-wave is slightly broad at the base. 3. This record was taken fifteen minutes later. The R-wave is distinctly abnormal. The heart was dilated and the contractions of the left ventricle were very atypical. 4. This record shows auricular fibrillation and an abnormal S-wave. The contractions of the left ventricle were now even more atypical. 5. This electrocardiogram was taken just prior to the time the ventricles began fibrillating. Note the rhythmical contractions of the auricles and complete heart block. There is now a broad and blunt R-wave.

months. During this time frequent electrocardiograms were taken. In only one instance did they yield electrocardiograms resembling those under discussion.

Herrick [16] recently reported a case of coronary occlusion which came to necropsy. An electrocardiogram taken of this patient a few

Fig. 12. — Dog 14. Shows an incision in the long axis of the interventricular septum which extends upward to the aortic valves. The cotton near the upper pole of this incision represents a traumatic communication between the ventricles. The right branch of the auriculoventricular bundle was undoubtedly cut. See electrocardiograms, Figures 13 to 15.

days prior to death was not of the type produced by the series of cases reported by Robinson, Oppenheimer and Rothschild, Carter and others. It is hard to conceive how the intraventricular conduction system escaped injury in the experimental hearts and in the clinical

16. Herrick, J. B.: Thrombosis of the Coronary Arteries, J. A. M. A. **72**: 387 (Feb. 8) 1919.

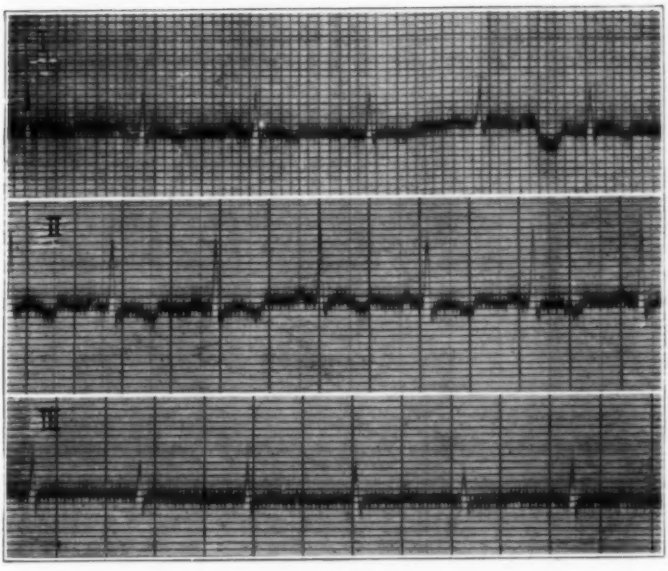

Fig. 13.—Dog 14. Initial electrocardiogram after heart was exposed.

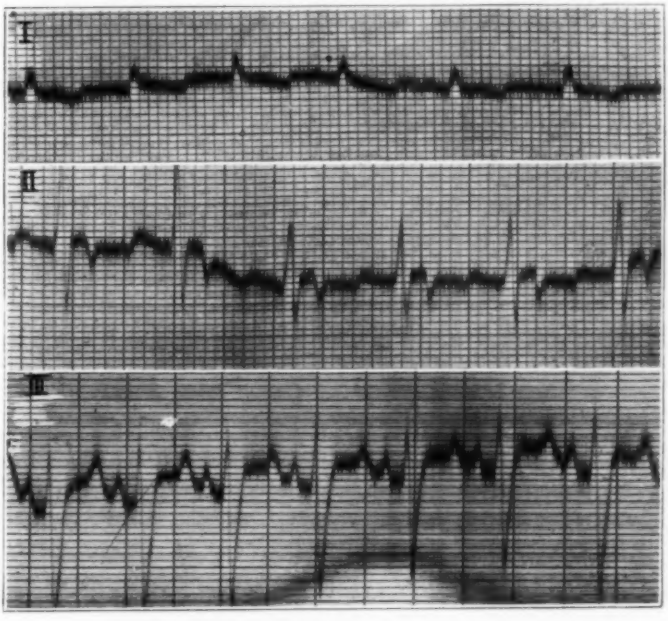

Fig. 14.—Dog 14. Electrocardiogram taken after a slit was made in the long axis of the interventricular septum (Fig. 12). There is a prominent S-wave in Lead III which is slightly broad at the base. This electrocardiogram indicates a preponderance of the left ventricle which in this instance is due to diminished contraction of the right ventricle.

case reported by Herrick with such an extensive fibrosis of the endocardial and subendocardial layers.

In the experiments covered by this report, normal electrocardiograms were obtained from hearts in which there were extensive incisions which extended through the endocardial and even the subendocardial layers. Later, however, when the hearts became dilated and the contraction modified the electrocardiograms were always of the abnormal type.

Fatigue of the heart muscle seemed to be an important factor, but not alone responsible for the abnormal electrocardiograms in our experiments. Experiments were done in which the branches of the

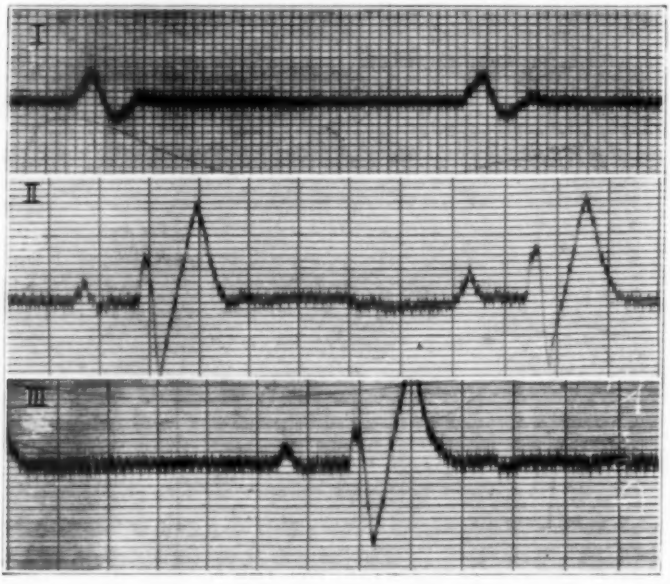

Fig. 15.—Dog 14. This electrocardiogram was taken at the time when the greater portion of the right ventricle was at a standstill and the contractions of the left ventricle atypical. Note the broad R- and S-waves. The P-R interval is increased.

coronary arteries were ligated and the heart forced to work in the partially closed hand of the experimenter until it became markedly dilated and finally went into ventricular fibrillation. Under these circumstances the heart often worked for more than an hour with the greater part of the coronary blood supply cut off from one ventricle. According to Hopkins and Fletcher,[12] and also Mines,[13] the conditions were ideal for an accumulation of acid metabolites in the musculature.

In none of these experiments did the dog give electrocardiograms with abnormal Q-R-S waves. If, however, in addition an incision was made in the endocardium these waves became atypical.

These observations would seem to indicate that there are at least two factors necessary for the production of the type of electrocardiogram under consideration, lesions of the intraventricular condition system and cardiac fatigue. Cardiac fatigue alone did not produce the atypical electrocardiogram. When, however, a defect was also made in the intraventricular conduction system this abnormal type of

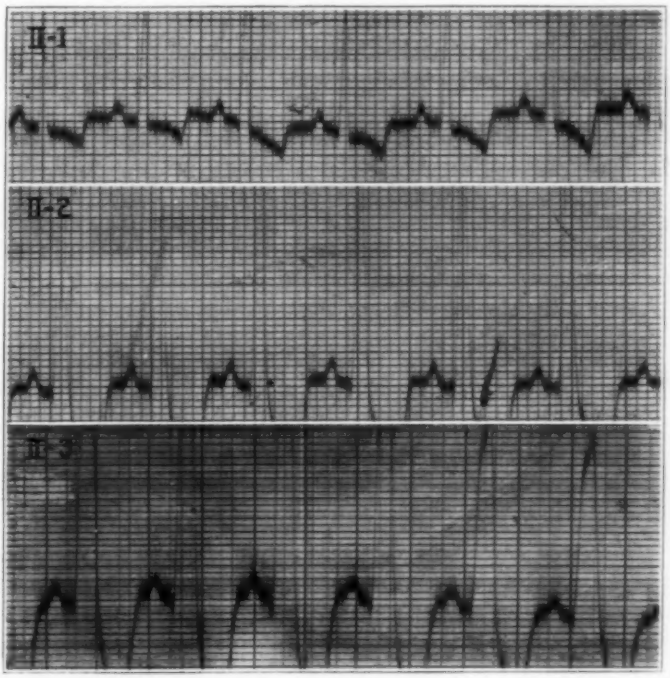

Fig. 16.—Dog 10. Lead II. 1. Initial electrocardiogram. 2. Record taken after transverse incisions were made in endocardium of left ventricle as shown by Figure 3. The height of the R-wave is increased and the base is slightly broad. The T-wave is more negative. The heart had begun to dilate. 3. Electrocardiogram taken after the ventricles had dilated and contractions become atypical. The R-wave is broad at the base and blunt at the peak.

electrocardiogram was always obtained. These results furnish a more satisfactory explanation of the abnormal Q-R-S waves of the clinical electrocardiogram. Patients are occasionally seen in whom these waves of the electrocardiogram become normal after rest and the adminis-

tration of digitalis. Some of these undoubtedly have permanent lesions of the intraventricular conduction system, as shown by Oppenheimer and Rothschild.[5] When, however, the physiologic processes of the heart are improved, the impulse may be able to reach all parts of the ventricles in normal time.

SUMMARY

A transverse slit was made in the interventricular septum just below the aortic valves in three dogs, cutting both branches of the auriculoventricular bundle. In one instance a short, shallow incision was made on the left ventricular side of the septum. In this case the left branch of the auriculoventricular bundle was divided. In one dog the right branch of the auriculoventricular bundle was divided by an incision in the long axis of the interventricular septum. In eleven dogs incisions were made in the endocardium of the left ventricle.

Electrocardiograms taken after one or both branches of the auriculoventricular bundle were divided showed some increase in the width of the R wave, and in some instances this wave was notched. In the one instance in which the right branch was cut there was an S wave in Lead III. Normal electrocardiograms were yielded by dogs in which there were extensive incisions in the endocardium of the left ventricle. Later as the ventricles became dilated and the contraction atypical the electrocardiograms became abnormal in all instances.

Cardiac fatigue alone was not responsible for the abnormal electrocardiograms in our experiments. Those hearts which were subjected to special strain by increasing the load and ligating the coronary arteries never produced an electrocardiogram with atypical Q-R-S waves. If, however, the endocardium of the left ventricle was incised these waves became abnormal. These experiments would seem to indicate that there are at least two factors necessary for the production of the abnormal electrocardiogram under consideration, lesions of the conduction system and cardiac fatigue.

It is a great pleasure to acknowledge my indebtedness to Dr. James B. Herrick for his many suggestions.

GASTRIC POLYPOSIS (PAPILLOMATOSIS)

REPORT OF A CASE WITH OPERATION AND PRESUMABLE CURE *

ERNEST S. DU BRAY, M.D.
SAN FRANCISCO

Confirmed cases of gastric polyposis are comparatively unusual, although reports of isolated cases of the disease have appeared from time to time in the literature for many years. Balfour [1] pointed out that in approximately 69,000 abdominal sections in the Mayo Clinic, 8,000 of which were for gastric lesions, his case was the first one of gastric polyposis that had been encountered. I cannot believe that this represents the true incidence of the disease, and in studying histories of gastric cases, it seems entirely plausible that some of the vague and obscure conditions of the stomach, masquerading under such diagnoses as chronic gastritis, gastric neuroses, etc., might, on further investigation, prove to be benign tumor conditions of the gastric wall. Ebstein [2] found fourteen cases of stomach polyps in 600 necropsies, and was able to collect reports of eight others from the earlier literature. Novak [3] recently recalled that it is from the French that much of our knowledge of these growths has come. Cruveilhier [4] has the credit of reporting the first undoubted case of adenoma of the stomach in 1849, and it is from this specimen that Brissaud [5] made his comprehensive study of benign gastric neoplasms.

There are several varieties of primary benign tumors of the stomach. Basch [6] states that the term polypus is merely descriptive and not histologic, since adenomas, fibromas, lipomas, myomas and papillomas may all form polypoid tumors, in which, sometimes, the connective tissue predominates, at other times, the glandular elements. The same author enumerates and discusses the types of benign tumors which have occurred in the stomach, mentioning mucous polypi (of which there are the two anatomic types, first distinguished by Menetrier [7]), adenomas, lymphadenomas, myomas, fibromas, lipomas, myxomas, osteomas and cysts. The last seven of these are extremely

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* From the Departments of Medicine and Surgery, University of California.

1. Balfour, D. C.: Surg., Gynec. & Obstet. **28**:465 (May) 1919.
2. Ebstein: Arch. f. Anat. u. Physiol., 1864.
3. Novak, E.: Polypoid Adenoma of the Stomach, J. A. M. A. **74**:871 (March 27) 1920.
4. Cruveilhier: Traité d'anatomie Pathologique, 1849, tome 2, livraisen 30, 2.
5. Brissaud: Arch. gén. de méd. **2**:257, 1885.
6. Basch, S.: Surg., Gynec. & Obst. **22**:165 (Feb.) 1916.
7. Menetrier, P.: Des Polyadénomes gastriques et leurs rapportes avec de cancer de l'estomac, Arch. de Physiol. norm. et path. **45**:32, 236, 1888.

rare tumors, and only a few scattered examples of each have been found in the literature. It is, therefore, safe to assume that of the primary benign tumors of the stomach the polyadenomas and papillomas form the important groups clinically.

Menetrier's [7] anatomic classification of the gastric polypoid conditions is still accepted and seems useful. He distinguishes two varieties, depending on whether the ducts or the fundus of the gastric glands are involved. When the ducts are principally involved, the tumor is distinctly lobulated, and cysts are more common, owing to the obstruction of the excretory ducts. On the other hand, when the alteration is chiefly in the fundus, there is little or no lobulation, and the cysts are either few in number or entirely absent. In this latter type, the polyps have a more uniform appearance and tubulation is less pronounced. To the first type he gives the name "polyadenomes polypeux" and to the second "polyadenomes en nappe." This latter type appears to be a very rare condition, and up to 1913, Meyer [8] states that only three authentic cases have been reported, one of them by Andral and the other two by Menetrier. Hayem [9] described a third type of tumor which he calls the Brunnerian adenoma. The glands are of the duodenal type and are thought to have their "anlage" in misplaced duodenal glands. Anatomically, the case I report conforms to the rare variety of Menetrier, the "polyadenomes en nappe;" however, the histologic structure is not that of an adenoma but of a papilloma. This finding is in keeping with Basch's observation that polypoid conditions in the stomach result from numerous types of microscopic tumor patterns.

The object of this paper is, therefore, to put on record another of these unusual cases of benign gastric tumors, which in this instance proved to be a polyposis of a papillomatous histologic structure. The patient was first seen in September, 1919, when I was working in the Medical Out-Patient Department of the University of California. This patient was observed over a period of about one month. Finally, a diagnosis of gastric carcinoma was made, which was concurred in by the roentgen-ray department, and the patient was then admitted to the hospital on Dr. Wallace I. Terry's surgical service.

REPORT OF CASE

H. S., white, male, single, aged 48, tailor by occupation, was admitted to the Medical Out-Patient Department of the University of California Hospital, Sept. 15, 1919, complaining of a feeling of pressure in the epigastrium, constipation and belching, especially at night.

8. Meyer, J.: Polyposis gastrica (Polyadenomata), J. A. M. A. **61**:1960 (Nov. 29) 1913.
9. Hayem: Presse méd. **2**:53, 1897.

Family History.—The patient's father died aged 79 of an unknown cause; the mother died during childbirth; one sister died at birth; one sister is living and well; one brother has stomach trouble. There is no history of cancer or tuberculosis in the family.

Past History.—The patient was born in Germany, emigrated at the age of 17 to the Middle West of the United States; when he was 25, he came to San Francisco, where he has made his home up to the present date. Besides the usual childhood diseases, he thinks he had the influenza about one year ago. There is no history of venereal diseases. In September, 1919, his tonsils were removed. His habits are regular; uses neither tea nor coffee in excess, but before his present illness he was accustomed to drink considerable beer; he has smoked a pipe excessively. During the past year his bowels have been constipated and there has been some nycturia once or twice at night.

Present Illness.—The patient was in good health until March, 1919, when he noted a sensation of fulness in the epigastrium, especially in the left side; the sensation was as if the stomach were full of gas and compelled him to belch frequently. This gas seems to press on the heart and cause palpitation, which is most marked at night. He does not suffer severe abdominal pain, but the fulness and pressure are present often before meals and are relieved at times for an hour after eating. His appetite has remained good. Frequently

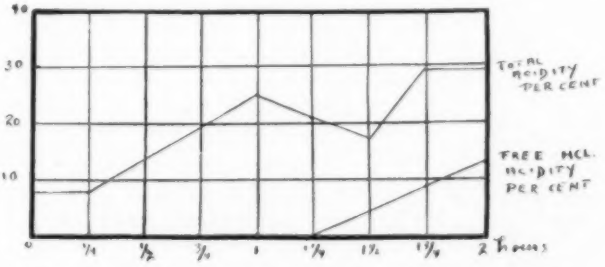

Fig. 1.—Fractional gastric analysis. Ewald test meal.

he has tried to vomit to relieve the distress, but without success. About one month ago he noted that his stools were black and tarry. He also thinks that he has been jaundiced several times since last March and that his constipation has been more marked since his present illness. He has noticed a slight shortness of breath for the past six weeks. There is no evidence of cough or night sweats. In March, 1919, his weight was 165 pounds; he thinks he has lost about 25 pounds since that time.

Physical Examination.—The patient is a fairly well developed and well nourished man of middle age, rather short and of heavy stature. Temperature, 98.2 F.; pulse, 74; respiration, 20. Height, 5 feet, 7 inches; ideal weight, 148 pounds; actual weight, 140 pounds. Blood pressure: systolic, 115; diastolic, 65. His hair shows the normal distribution with beginning baldness over the top of the head. The skin is dry with a moderate amount of pigmentation, particularly over the face. The eyes are normally set, the pupils equal, rather small and react to light and accommodation; the extra-ocular movements are good. The nose and ears on superficial examination are normal. The tongue is moderately coated and protrudes in the midline without tremor. The teeth are stained, probably from tobacco. The tonsils have been removed recently. Neither general nor local glandular enlargement is found. The chest is negative. The heart is within the normal limits as regards size, shape and position; the apex beat is distinct in the fifth left space, 10 cm. from the

midsternal line; the sounds are regular and rather distant; there are no murmurs; the pulmonary second sound is equal to the aortic second sound; the radial pulses are equal and regular. The vessel wall is not felt. The costal angle is rather wide and there is moderate depression of the epigastrium. The abdominal walls are lax and soft, and there are no abnormal masses felt and no visible peristalsis. On rather deep pressure in the epigastrium there is slight tenderness which is not definitely localized. No tumor mass can be palpated. There is also slight tenderness to deep palpation in the left lower quadrant. The extremities are negative and the deep reflexes are equal and active throughout. Rectal examination shows no hemorrhoids, external or internal; the sphincter is normal. No perirectal glands are palpable. The prostate is small and soft and the median fissure is well marked.

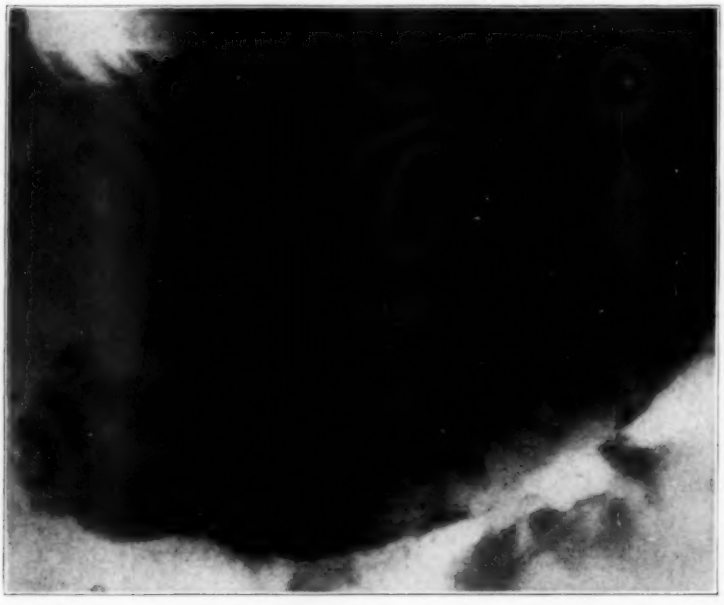

Fig. 2.—Roentgenogram picture showing large, complete filling defect in the greater curvature after six hours. In the lower portion of this defect there is only slight mottling, with the finger-print pattern described as the typical lesion of benign gastric tumors.

Laboratory Findings.—Wassermann blood reaction is negative. Fractional gastric analysis is shown in Figure 1. Residue from fasting stomach, 150 c.c. Occult blood test positive (benzidine). Many pus cells and much mucus present.

Urine examination of single specimen: Specific gravity, 1.020; urine clear, reaction acid; albumin and sugar negative; sediment negative.

Blood examination: Hemoglobin, 90 per cent. (Sahli); red blood cells, 4,800,000; white blood cells, 8,600. Differential count: polymorphonuclears, 65 per cent.; small mononuclears, 28 per cent.; large mononuclears, 7 per cent.

Stool examination (meat-free diet): Benzidine test positive.

The results of roentgen-ray examination are shown in Figure 2.

Fluoroscopic Examination: Lung fields show a wide aortic arch and a slightly enlarged heart. The diaphragm moves well and equally on the two sides. No delay is noted in the esophagus. The stomach tone is good, it is readily movable and its position is normal. There is no residue after six hours. There is a large defect in the greater curvature. The waves start high up on both curvatures and pass clear through over the lesser curvature, but fade out in the region of the defect in the greater curvature to reappear again below. The pyloric and duodenal caps are normal. The previous meal is found in the terminal ileum, cecum and the transverse colon, all of which are freely movable, separable and not tender.

Diagnosis.—Carcinoma of the stomach—Dr. Ruggles.

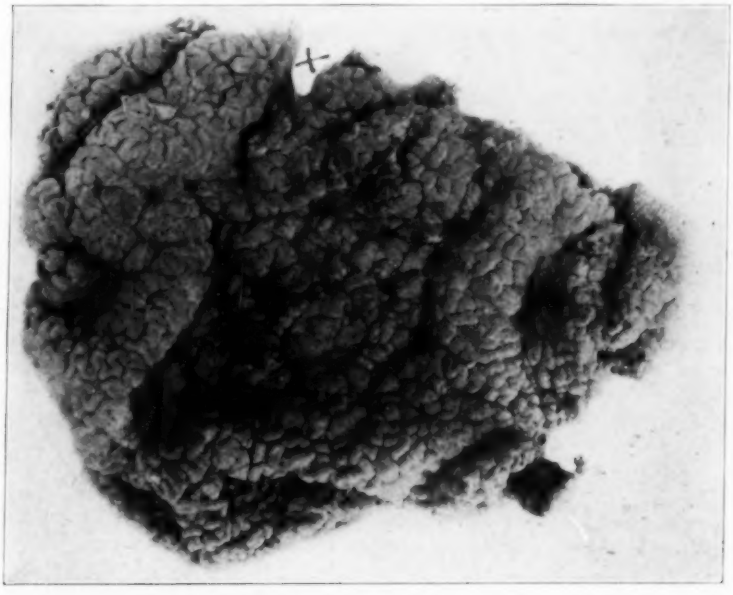

Fig. 3.—Specimen removed at operation.

CLINICAL SUMMARY

A tailor, aged 48, with a history of epigastric pain and belching; family history and past history unimportant, has been ill about eight months, with history of loss of weight. The finding of practically negative physical examination, except for epigastric tenderness, points to the possibility of a stomach condition. The absence of free hydrochloric acid in the stomach juice and the finding of occult blood in the stools together with the fluoroscopic picture, which revealed a large, clean cut defect in the greater curvature, indicated a diagnosis of gastric carcinoma. The patient was then sent to the surgical service of the hospital.

Operation.—Exploratory laparotomy, Dr. Wallace I. Terry, Oct. 29, 1919: A longitudinal incision was made through the right rectus muscle, extending from the costal margin to 3 cm. below the umbilicus. The abdomen was opened in the usual way. The foramen of Winslow was open. The abdominal contents were apparently negative. The stomach externally showed no patho-

logic signs, except that there were a few soft lymph glands near the attachment of the great omentum to the greater curvature of the stomach. At the greater curvature of the stomach, however, a mass could be palpated within, which was readily movable under the finger and not infiltrating the muscular or peritoneal coats. The impression conveyed was that of a large doughy tumor about 8 cm. in diameter. The presence of such a tumor immediately threw doubt on the preoperative clinical diagnosis. A frozen section of one of the glands above mentioned was made and no carcinomatous changes could be demonstrated in this tissue. The stomach was therefore opened to confirm the impression that a polypoid condition of the gastric wall was present; this proved to be correct. After closely applying abdominal tapes to the gastric wall the stomach was opened, and then grasping the periphery of the tumor with a couple of Allis forceps, it was excised. The great omentum had to be detached from this portion of the greater curvature. The wound was sewed together after the manner of Finney with mattress sutures of iodized catgut, running stitch, with a reinforcing stitch over this. So great a portion of the greater curvature and walls of the stomach had been removed, it was feared that an hour-glass stomach would result; consequently, a gastroenterostomy was also performed. The cardiac end of the stomach was laid next to the proximal loop of the jejunum by means of the Allis forceps, the stomach being brought through the opening in the mesocolon, which had been turned up over it. The stomach was then grasped with an intestinal clamp and the jejunum with a Moynihan clamp. There was some difficulty in approximating the two pieces of bowel on account of the small amount of stomach that could be seized through the rest of the mesocolon. They were, however, finally joined by an approximating stitch of catgut and the two portions of bowel cut longitudinally, and the opposing and remote edges approximated by means of a Connell stitch. A last reinforcing stitch was applied to the now closed stoma and the jejunum fixed to the hole in the mesocolon by means of three stitches of iodized catgut. The abdomen was closed in the usual manner.

REPORT OF THE SURGICAL PATHOLOGY LABORATORY OF THE UNIVERSITY OF CALIFORNIA

Case of Dr. W. I. Terry, Oct. 31, 1919. H. S., S. 19, 792.

Gross Pathology.—The specimen (Fig. 3) consists of a portion of stomach wall measuring 13 by 10 cm. The peritoneal surface does not show anything grossly abnormal, except some clamp marks and a few hemorrhagic stains. The mucosal surface presents a tumor mass measuring 11 by 10 cm., surrounded by a minimum of what in the gross appears to be quite normal stomach mucosa. The superficial picture shows closely packed papillomatous growths, thrown into folds, which resemble very much in color and contour brain cortex. It is relatively easy to separate the various convolutions (that is, the individual papillomas) clear to their bases. There can be traced a gradual increase from the apparently normal mucosa to the pronounced papillomatous outgrowths. No gross evidence of necrosis can be made out in any of the papillomas, although there is a slight amount of hemorrhagic discoloration in the tips of some of them. On section the gross picture does not show any increase in the muscular or connective tissue elements of the stomach wall proper. There is no gross evidence of any extension downward into the layers beneath the mucosa nor are there any other suggestions of malignant change. The mucosa displays a decided thickening in places as much as 0.5 cm. Gross impression: Papillomatosis of the stomach (benign).

Microscopic Pathology (Figs. 4 and 5).—Duplicate sections of the tumor were stained respectively with the routine hematoxylin and eosin, for muscle and connective tissue with Van Giesen's, for connective tissue by Mallory's method, and for elastic tissue by Weigert's stain. Sections show a picture of stomach mucosa displaying a gradual increase from normal thickness to

tremendous hypertrophy and thickening in a definitely papillomatous arrangement. The general picture of this hypertrophy is that of gastric glands. The tubular glands are greatly elongated, show a marked corkscrew type of growth and many branchings. The mucosal cells are invariably columnar in type and in many places, especially in the depths, show wonderfully clear differentiation from each other; the distinction between the chief and parietal

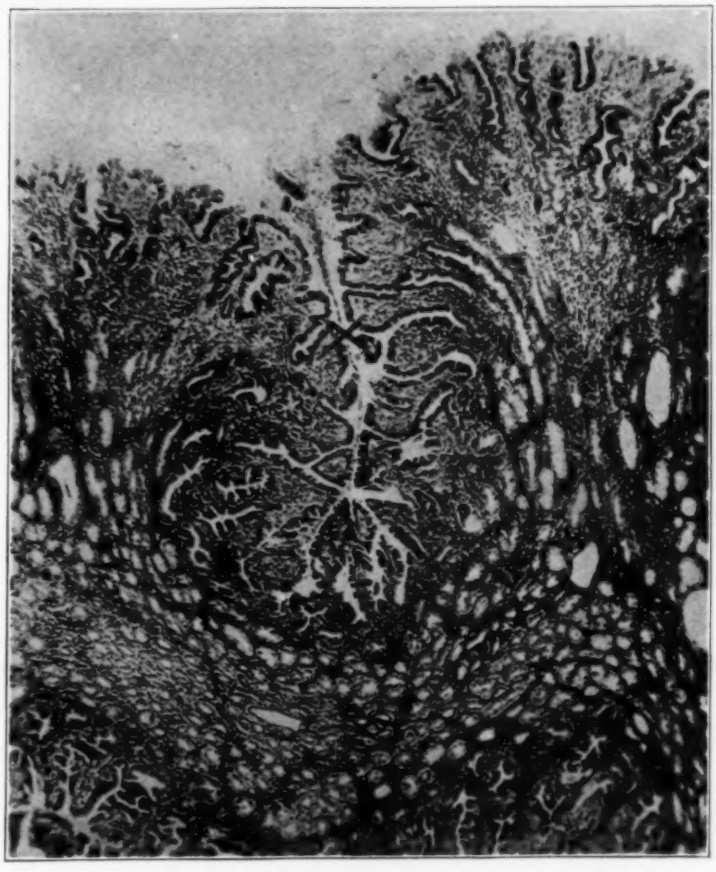

Fig. 4.—Photomicrograph, low power. Block taken from point X on Figure 3.

cells is striking. Occasionally, one sees some atypical nuclei which are definitely shown to be the result of degeneration instead of mitosis. The stroma is minimal in amount. The sections stained to show muscle and connective tissue by Van Giesen's method show the stroma of the papillomatous outgrowths to be connective tissue and to contain no muscle. The connective tissue is verified by Mallory's stain. Weigert's stain brings out no trace of elastic fibers within these structures. These special stains, more especially

the Mallory's, demonstrate the fact that there is no breaking up of the basal membrane of the glandular structures and show it to be everywhere intact. The stroma shows in places some edema, some infiltration with round cells, plasmalike cells, red blood cells and an occasional polymorphonuclear. The stomach musculature proper is everywhere normal in appearance.

Diagnosis: Papillomatosis of the stomach—Granville S. Delamere, M.A.

Clinical Course.—The postoperative course in the hospital was uneventful. November, 1919, a gastro-intestinal series following a bismuth meal showed

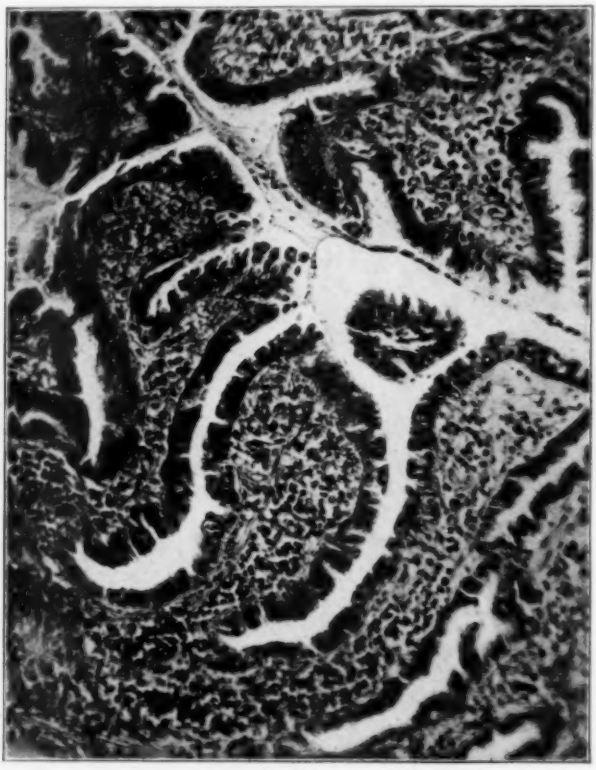

Fig. 5.—Photomicrograph, high power. Field X on Figure 4.

a large postoperative defect in the middle third of the stomach, the pyloric and duodenal caps normal, and the gastro-enterostomy apparently working well. There was some delay in the duodenojejunal juncture with some moderate amount of reversed peristalsis. Conclusion: Postoperative condition.

Nov. 22, 1919, the patient was discharged from the surgical service and advised to report regularly to the Medical Out-Patient Department. Dec. 11, 1919, in the Surgical Out-Patient Department a sigmoidoscopic examination was done (Dr. Hyman). The tube was easily introduced a distance of 23 cm. and by means of distention with gas the bowel wall was visible for 5 cm. farther. The picture was entirely normal.

Feb. 19, 1920, a fluoroscopic examination was made. Gastro-enterostomy was seen to be functioning well; meal also left stomach through the pylorus; postoperative defect seen in the median third of the stomach, but no evidence of obstruction.

March 20, 1920: The patient's course in the Medical Out-Patient Department has been uneventful. Patient has gained twenty pounds in weight and is feeling well.

ETIOLOGY

Nothing definite is known about the cause of gastric polyposis. Its frequent association with other diseases, such as gastritis, hypertensive cardiovascular disease, and syphilis has led to interesting speculations.

SYMPTOMATOLOGY

There is nothing pathognomonic in the symptoms of this condition, although there are, at times, certain suggestive evidences of the disease. The fact that many of the cases are discovered at necropsy, when the existence of ante mortem gastric disease is not suspected, demonstrates its frequent silent nature. Clinical manifestations, however, when present, depend principally on several factors, namely, the size, location and nature of the growth, and particularly its relation to the pyloric region of the stomach. It is found more commonly in men past middle age.

Nonpedunculated, broad based masses on the greater curvature may remain symptomless or more commonly produce vague, indefinite symptoms of an epigastric dragging sensation, followed by a desire to belch and eventually to vomit. The onset of such disturbances does not appear to have any relation to meals, since it is as often present before meals as after or between meals. Meyer, in his careful study, calls particular attention to the type of vomitus which he regards as of great significance. The vomitus he describes contains an excessive amount of mucus of an egg-white appearance and in his case a small benign polyp (adenoma) was recovered, which enabled him to make the pre-operative diagnosis. Further, it is noted in these cases, that there is usually absence or greatly reduced amount of free hydrochloric acid, in other words, an achylia gastrica or hypoacidity.

Pedunculated and less commonly nonpedunculated growths, situated on the stomach wall near the pylorus may prolapse and present the typical clinical picture of pyloric obstruction, partial or complete. Wade [10] reports a case which is a spectacular example of this variety.

Hemorrhage, slow and long unrecognized, is a frequent finding in these cases and is due to the great vascularity of these tumors of the mucous membrane. This accounts for the usual finding of occult blood in the fasting contents of the stomach and in the stools.

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10. Wade, H.: Intussusception of the Stomach and Duodenum Due to Gastric Polyposis, Surg., Gynec. & Obstet. **17**:184, 1913.

The appetite is variable; perhaps, more often it remains good and for this reason is in marked contrast to anorexia so prevalent in the cases of gastric malignant disease.

The general nutrition, weight and blood picture are also variable, but, as a rule, they are less altered than in the carcinoma cases, particularly when pyloric obstruction has occurred. Careful observations in regard to these latter conditions are among the most helpful adjuncts in the final estimation of a case.

It may be said, in general, that palpable masses are more common in these cases when the growth is large and located near the pylorus, but it is surprising in how many cases nothing abnormal can be determined by abdominal palpation. Intense pain in the epigastrium is recorded in an occasional case and is in some instances the symptom which first brings the patient to the physician.

The duration of the history in these cases is, at times, significant; the fact that definite symptoms may have been present for a long time without apparent progression and with little or no change in the patient's general health is strong evidence against the presence of malignant disease, and should suggest the possibility of a benign tumor condition.

DIAGNOSIS, WITH SPECIAL REFERENCE TO ROENTGEN RAY

Gastric polyposis, as has been indicated, besides being an accidental finding at necropsy, may also be discovered in the course of surgical procedures. The early accurate roentgenographic description by Carman in Meyer's case of gastric polyposis, in which a fairly characteristic picture of the disease was first outlined, might seem to have indicated that definite roentgenographic manifestations were always to be expected in such cases. Unfortunately, this has not proved to be the case, since we know now that gastric polyposis may present roentgenographic evidence so similar to that found in cases of gastric carcinoma that not even an expert roentgenologist can differentiate the two conditions. The interpretation of the findings in benign polypoid tumors from the presence of gastric carcinoma is the prime difficulty. Many recorded instances exemplify this, and the case reported above is a typical example in which roentgen-ray interpretation was misleading both to the internist and to the roentgenologist, because the manifestations conformed more to the usual picture seen in carcinoma of the stomach than to the rarer condition of gastric polyposis. In the two cases of polyposis reported by Friedenwald and Finney,[11] notwithstanding the roentgen-ray studies, the diagnosis in both instances was gastric carcinoma.

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11. Friedenwald, J., and Finney, J. M. T.: Gastric Polyposis, Am. J. M. Sc. **154**:683 (Nov.) 1917.

It would appear that when the typically mottled, fingerprint, filling defect is present in the stomach, in conjunction with other evidence in the history, physical examination and laboratory findings pointing to benign gastric tumor, a diagnosis of gastric polyposis may be justified, but, conversely, it must be remembered that roentgenograms strongly suggesting malignant new growths may prove to be benign tumors.

In view of the foregoing statements, it appears that roentgen-ray studies of the stomach have strengthened the surgeon's position in regard to the advisability of exploratory laparotomy in some of the presumably advanced cases of gastric carcinoma, since in rare instances the finding of a benign tumor may be a surprise to the surgeon and a "new lease of life" to the patient. The roentgen ray affords valuable evidence in some cases and misleading evidence in others. Carman and Miller [12] state that in view of the limited number of these cases our roentgen-ray experience is still necessarily meager, and, therefore, it is not possible to lay down hard and fast rules concerning the roentgen manifestations.

Besides the roentgen-ray studies of the stomach, other helpful methods of investigation should include a careful history, a thorough general physical examination, and numerous laboratory tests. When the combined results of these procedures are evaluated and combined with the roentgen-ray findings, we have at our command the methods which offer the greatest expectation of a correct diagnosis.[13]

SURGERY

Excision of the tumor mass and such reconstructive measures as may seem indicated in the individual case offer the greatest hope for satisfactory results in cases of gastric polyposis.

SUMMARY

A case of gastric polyposis of the papillomatous type is reported in which the clinical and roentgen-ray diagnosis was carcinoma of the stomach. Exploratory laparotomy revealed a broad based benign tumor situated in the greater curvature of the stomach; this tumor was excised and following this the patient has had an uneventful convalescence and six months later has had no recurrence of his former symptoms.

I wish to take this opportunity to express my thanks to members of the various departments of the University of California Hospital, through whose cooperation and efforts the study of this case has been made possible.

12. Carman, R. D., and Miller, A.: The Roentgen Diagnosis of Diseases of the Alimentary Canal, Philadelphia, W. B. Saunders Company, 1917.

13. For a complete bibliography of the older literature see Friedenwald and Finney (Footnote 11).

CLINICAL AND ELECTROCARDIOGRAPHIC OBSERVATIONS ON INVERSION AND OTHER ANOMALIES OF THE P WAVE*

WALTER W. HAMBURGER, M.D.
CHICAGO

Interest regarding the diagnostic and prognostic significance of inversion and other anomalies of the P wave of the human electrocardiogram continues to grow. Much of this interest centers around the problem of interpretation. Likewise, the frequency of this finding seems to increase, although this is probably the result of attention being focused on it. During the past six months, approximately three hundred curves have been taken in the electrocardiographic laboratory of the Michael Reese Hospital, of which fully 20 per cent. have shown inversion of the P wave, usually in Lead III. In view of this frequency, and because exact knowledge regarding its significance is meager, it seemed worth while to analyze the curves showing this phenomenon and summarize the findings thus obtained.

It is generally accepted that the physiologic auricular complex—P—is an upright wave which results from auricular systole and "expresses the origin of the heart beat at the normal site of impulse formation and the passage of the wave through the whole of the auricular tissue in definite directions."[1] According to Hoffman,[2] the P wave accompanies conduction over the auricle. Variations in this wave, therefore, in height, length, direction, configuration, etc., are dependent on deviations from the normal, either at the site of impulse formation (pacemaker) or of the conduction paths through the auricle. Certain factors responsible for these variations have already been described.

Einthoven[3] demonstrated that vagal stimulation may so reduce the height of the P wave that it becomes diphasic. Thomas Lewis[4] showed that a change in the shape of P indicated a change in the location of

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* From the Electrocardiographic Laboratory of the Michael Reese Hospital, Chicago.

* Read at the Meeting of the American Society for Clinical Investigation, Atlantic City, N. J., May 3, 1920.

1. Lewis, T.: Clinical Electrocardiography, Ed. 2, 1918, London.
2. Hoffman: Die Electrocardiographie, 1914, Weisbaden, Bergman.
3. Einthoven, W.: Weiteres über das Elektrokardiogram, Arch. f. d. ges. Physiol. **122**:540, 1908.
4. Lewis, T.: Galvanometric Curves Yielded by Cardiac Beats Generated in Various Areas of the Auricular Musculature, The Pacemaker of the Heart, Heart, **11**:2, 1910.

the pacemaker. He found, for Lead II, that when the impulse originated in the upper portion of the auricle, the P wave was upright. When the impulse originated in the median part of the auricle, P approached the isoelectric state. When it originated in the lower part of the auricle, P was inverted.

Ritchie,[5] v. Hoesslin,[6] Einthoven, Fahr and deWaart [7] ascribed the inversion of P, which they observed after vagal stimulation, to some change in the pacemaker, or to some change in the path of contraction wave over the auricle.

Hering [8] showed that in the first lead, the size and direction of P may not be affected markedly, even when the impulse originated in the A-V node.

Goddard [9] believed that inversion of P occurred in Lead III only, that it occurs at all ages, and that the size and shape of P may vary in the same curve, that an inverted P in one record may appear upright in a later tracing, and vice versa.

Wilson [10] reported three cases showing changes in the location of the pacemaker associated with deep respiration, and believed that the changes might be divided, first into migration of the pacemaker within the sino-auricular node or its immediate neighborhood, and second, migration of the pacemaker from the sino-auricular to the A-V node.

Carter and Wedd [11] summarized their findings as follows: "We believe we may divide our series of inverted P waves seen in Lead III into two distinct groups. One in which there may be a variation of the site of the pacemaker in its relation to Lead III, conspicuously under the influence of the vagi and digitalis. Second, in which the relation of the pacemaker to Lead III is such that a constantly negative P 3 is present, utterly uninfluenced by atropin, vagal pressure of

5. Ritchie, W. F.: Action of the Vagus on the Human Heart, Quart. J. Med. **6**:47, 1912.

6. v. Hoesslin, H.: Beobachtungen über den Einfluss der Vagus auf dass Menschlichen Herz, Deutsch. Arch. f. klin. Med. **113**:537, 1914.

7. Einthoven, Fahr and deWaart: Ueber die Richtung und dier manifeste Groesse der Potential Schwankungen im menschlichen Herzen und über den Einfluss der Herzlage auf die Form des Electrokardiograms, Arch. f. d. ges. Physiol. **100**:275, 1913.

8. Hering, H.: Rythmische vorhofstach systolie und Pulsus irregularis perpetuem, München. med. Wchnschr. **111**:2057, 1914.

9. Goddard, C. H.: Changes in the P Wave of the Human Electrocardiogram, Arch. Int. Med. **16**:633 (Nov.) 1915.

10. Wilson, F. N.: Three Cases Showing Changes in the Location of the Cardiac Pacemaker Associated with Respiration, Arch. Int. Med. **16**:86 (July) 1915.

11. Carter and Wedd: Observations on the Occurrence of Inverted and Diphasic P Waves in Lead III of the Human Electrocardiogram, Arch. Int. Med. **23**:1 (Jan.) 1919.

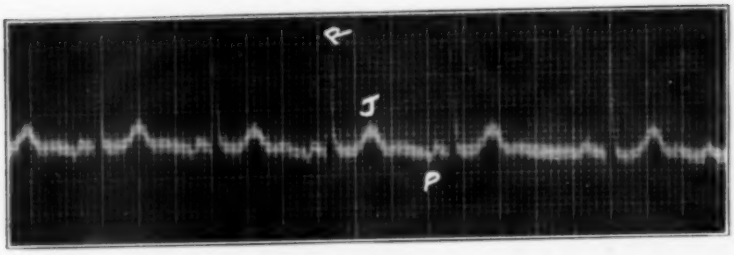

Figure 1

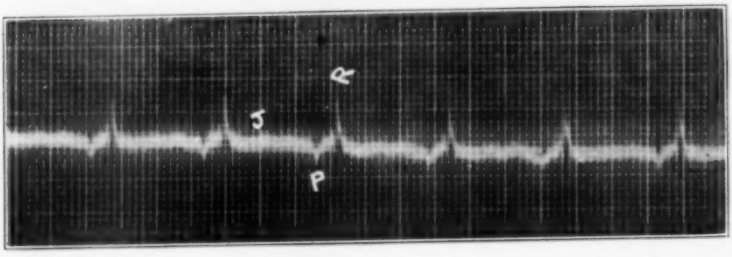

Figure 2

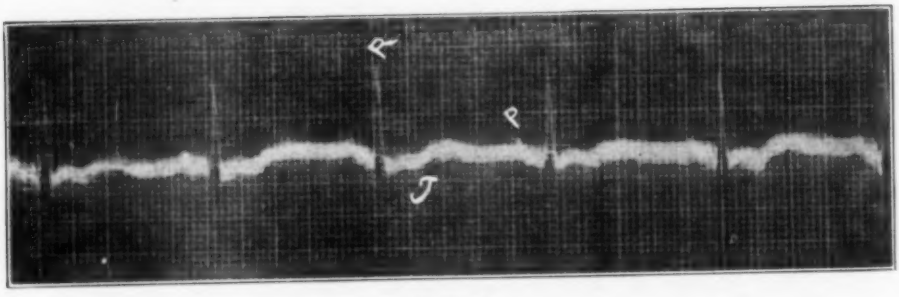

Figure 3

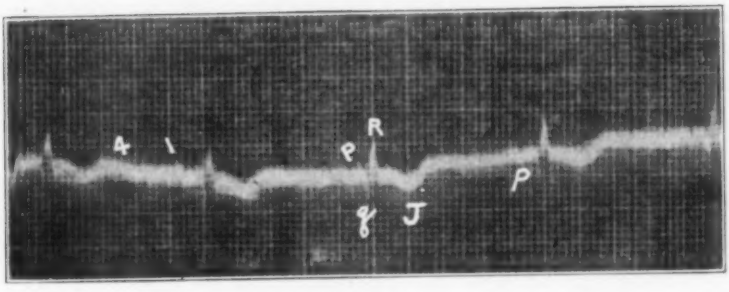

Figure 4

digitalis, due probably to a change in the muscle balance altering the relation of the pacemaker to the axis, or to the distribution of the vagal fibers."

White reported one case showing migration of the pacemaker within the sino-auricular node.

Present knowledge regarding variations of the P wave, therefore, may be summarized as follows:

1. Vagal stimulation (vagus pressure) reduces the height of the P wave, makes it diphasic and causes inversion.

2. The P wave becomes isoelectric when the impulse originates in the median portion of the auricle; inverted when it originates in the lower part of the auricle.

3. Deep respiration causes changes in the appearance of an upright P, flatting and inversion with forced expiration, with a return to upright upon inspiration.

4. Digitalis may cause inversion of P.

5. Atropin may cause an inverted P to become upright.

6. Persistently inverted P waves may occur, uninfluenced by vagus pressure, digitalis or atropin.

7. Inversion of P is usually associated with a decrease in the P-R interval, sometimes with slowing of rhythm, or the setting up of A-V rhythm.

8. Inversion of P is believed to express migration of the pacemaker of the heart from its normal position in the sino-auricular node to some ectopic point lower down in the auricle, even to the auriculoventricular node.

The following cases comprise the ones on which this report is based. For the sake of brevity only the salient points in connection with these cases are given.

REPORT OF CASES

Case 1.—M. S., aged 12 years. Clinical diagnosis: Sinus arrhythmia. Lead II: Wave at first inverted (P-R interval 0.12); later becomes upright (P-R 0.14), with slowing of pulse (Fig. 1). Lead III: Permanently inverted (P-R 0.12) (Fig. 2).

Case 2.—S. W. S., aged 55 years. Clinical diagnosis: Pyonephrosis. Lead III: Recumbent posture; P wave upright (P-R 0.16) T inverted (Fig. 3). Lead III: Upright position; P wave inverted (P-R 0.12). T inverted (Fig. 4).

Case 3.—A. W., aged 60 years. Clinical diagnosis: Fibrous myocarditis. Lead II: P irregularly inverted; auricular and ventricular extrasystoles (Fig. 5). Lead III: P inverted at first (P-R 0.16); later upright (P-R 0.20) (Fig. 6).

Case 4.—M. M. H., aged 58 years. Clinical diagnosis: Fibrous myocarditis. Lead I: P inconstant and diphasic (P-R 0.16) (Fig. 7). Lead III: P bifurcate (P-R 0.20) (Fig. 8).

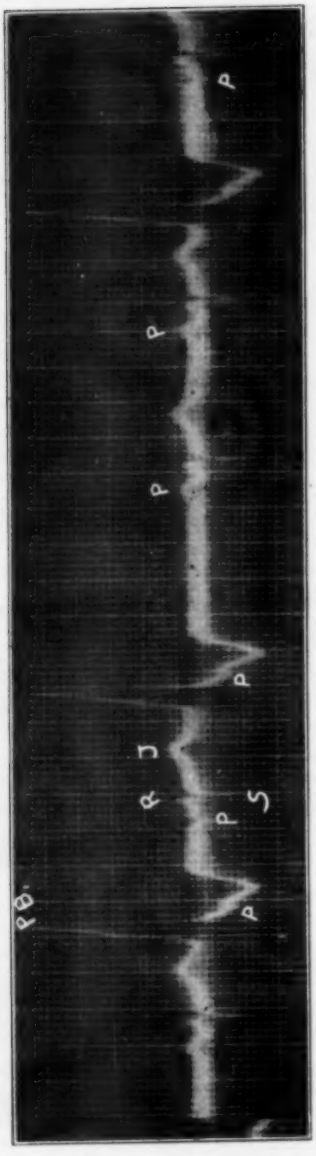

Figure 5

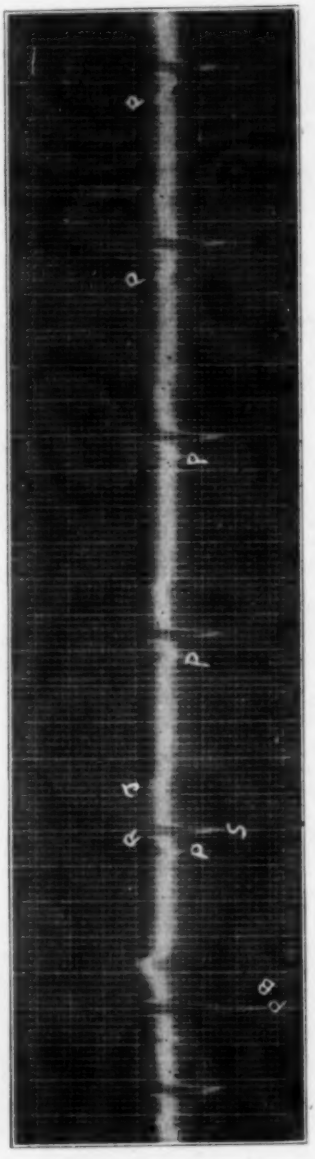

Figure 6

CASE 5.—B. B., aged 11 years. Clinical diagnosis: Subacute myocarditis. Lead III: P diphasic; notching of P-R interval (P-R 0.12) (Fig. 9).

CASE 6.—Miss M. L., aged 40 years. Clinical diagnosis: Fibrous myocarditis. Lead III: P inverted and wide (P-R 0.16) (Fig. 10).

CASE 7.—Miss G. Clinical diagnosis: Postscarlatina and diphtheria; myocardial insufficiency. Lead III: P inverted with exception of a single upright wave. Inverted P bifurcated and wide; later upright and narrow. Inverted P (P-R 0.16); upright P (P-R 0.16) (Fig. 11).

CASE 8.—Mrs. C. Clinical diagnosis: Postinfluenzal myocardial insufficiency. Lead III: P inverted, narrow and shallow (P-R 0.16) (Fig. 12).

CASE 9.—Dr. H. Clinical diagnosis: Myocardial weakness. Lead III: P inverted, varying depths. Muscular tremors (P-R 0.16) (Fig. 13).

CASE 10.—Mr. B., aged 52 years. Clinical diagnosis: Hypertension. Lead III: Diphasic P (P-R 0.16) (Fig. 14).

CASE 11.—Mr. B. F., aged 53 years. Clinical diagnosis: Fibrous myocarditis, myocardial insufficiency, angina pectoris. Lead III: P diphasic bifurcated (P-R 0.16) (Fig. 15).

CASE 12.—Miss W. A., aged 40 years. Clinical diagnosis: Postinfectious myocardial weakness. Lead III: P diphasic (P-R 0.16) (Fig. 16).

CASE 13.—Mr. G., aged 50 years. Clinical diagnosis: Visceral syphilis; Wassermann + + +. Lead III: P upright and wide (P-R 0.18); auricular extrasystoles (Fig. 17). Lead III: After 1/60 grain atropin, P inverted and narrow (P-R 0.11), auricular extrasystoles (Fig. 18).

CASE 14.—Mr. D. Y., aged 56 years. Clinical diagnosis: Myocarditis. Lead III: P inverted (P-R 0.16).

CASE 15.—Miss N., aged 26 years. Clinical diagnosis: Myocarditis, postinfluenzal. Lead III: P inverted (P-R 0.14); auricular extrasystoles.

CASE 16.—Dr. B., aged 34 years. Clinical diagnosis: Myocarditis, postinfluenzal, auricular extrasystoles, dropped beats. Leads II and III: P inverted (P-R 0.32 to 0.36).

CASE 17.—Mr. C. W., aged 35 years. Clinical diagnosis: Myocarditis, postinfluenzal. Leads II and III: P inverted (P-R 0.12); ventricular extrasystoles.

CASE 18.—Mr. R., aged 30 years. Clinical diagnosis: Myocarditis, postinfluenzal, auricular extrasystoles, pulsus bigeminus. Leads I, II and III: Inverted P.

An analysis of these cases brings out the following facts.

All ages are represented, the youngest patient was 11 years, the oldest 60. Clinically, in a majority of instances, some evidence of myocardial insufficiency could be demonstrated, in degrees varying from slight heart muscle involvement to grave forms of cardiac embarrassment. Many are acute or subacute and transitory, or postinfectious (influenzal) (Streptococcus) disappearing within a varying length of time after the etiologic infection has subsided. In six cases in which definite myocardial involvement could be diagnosed, inversion of P was associated with arrhythmia, auricular (occasionally ventricular) extrasystoles. Several cases were found in neurotic (vagotonic?) individuals, following slight acute respiratory infections, excessive smoking, prolonged nervous or mental strain, chronic gallbladder or kidney infections. Two cases were associated with disturbances of respiration, "difficulty in taking a deep breath," and with

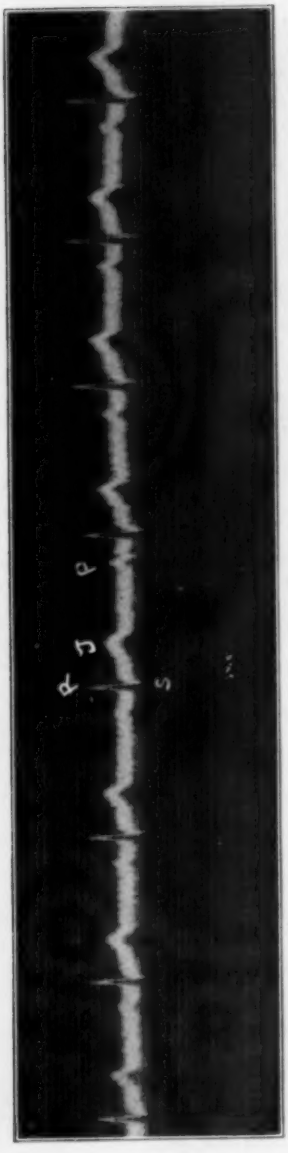

Figure 7

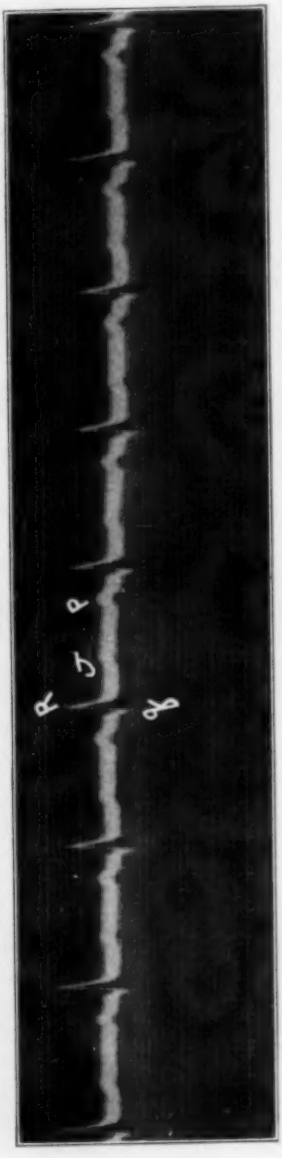

Figure 8

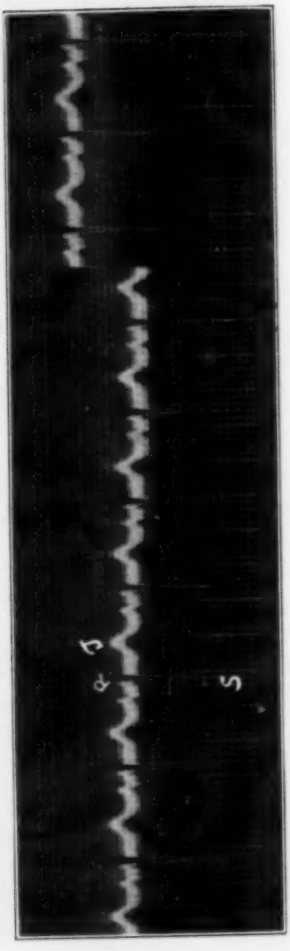

Figure 9

tachycardia on exercise. Three cases of inversion become upright after atropin; one upright P (Case 13) became inverted after atropin. In one case (Case 2) an upright P with patient recumbent, became inverted when patient sat up. In the majority of cases, inversion was present in the third lead only; in five cases it was present in Leads II and III; in one case it was present in all three leads.

Four cases showed regular or irregular alteration of inverted and upright P; in these cases, P-R of the inverted waves was shorter (average 0.04) than that of the upright waves. Width and depth of the inverted waves often was different from the upright waves; at times being narrow and shallow, and at other times being wide and deep. Five cases showed diphasic waves; four showed notched or bifurcated waves.

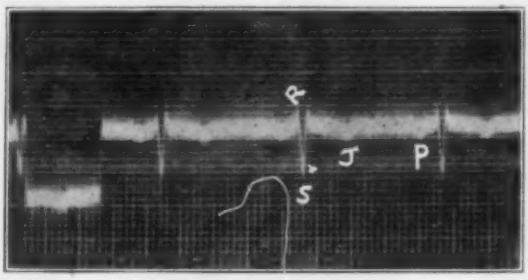

Figure 10

DISCUSSION

Assuming that the various anomalies in the P summit cited above are evidence of a migration of the site of impulse formation (pacemaker) and a consequent disturbance in the passage of the conduction wave through the auricle, one must conclude that such variation is very frequent (20 per cent.), extremely bizarre, and dependent on a number of factors, difficult to correlate. The frequency of inversion in Lead III, and the infrequency in other leads, suggests, as in inversion of T, that this may be a phenomenon of little significance. Against this view is the fact that in a majority of the cases some degree of myocardial weakness could be demonstrated clinically, as well as the fact that most of the patients suffered, or had suffered with acute or chronic infectious processes, factors of etiologic importance in cardiac pathology. Cases in this series in which inversion is associated with auricular extrasystoles gave unquestioned evidence of grave myocardial involvement, so that one may feel justified in stating, that migration of the pacemaker *with* auricular premature contractions is, in the majority of instances, to be interpreted as electrocardiograph evidence

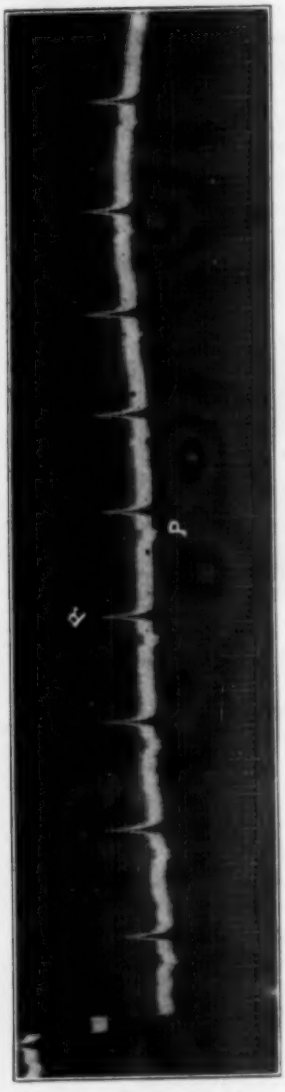

Figure 11

Figure 12

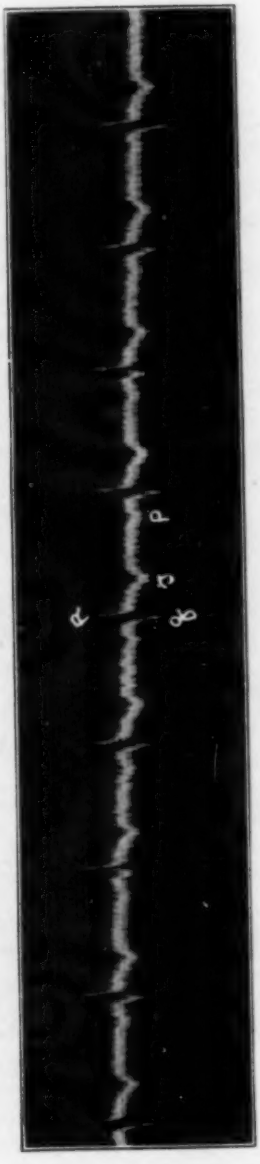

Figure 13

Figure 14

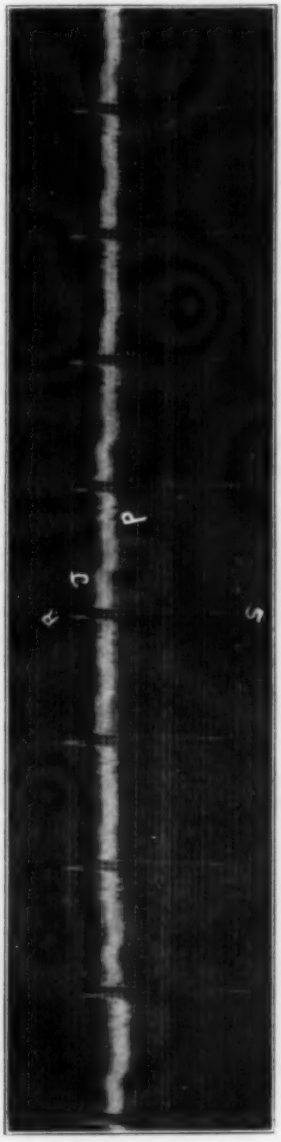

Figure 15

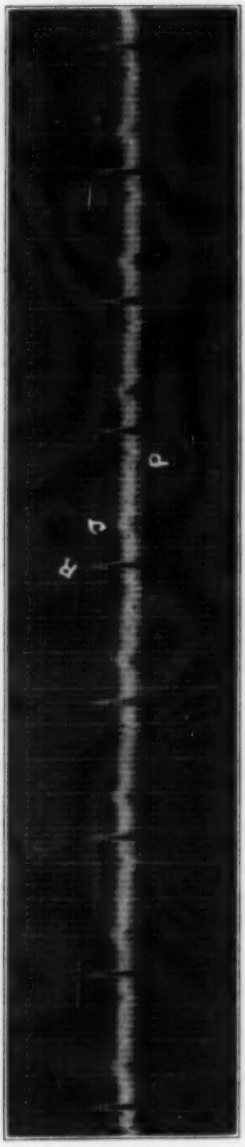

Figure 16

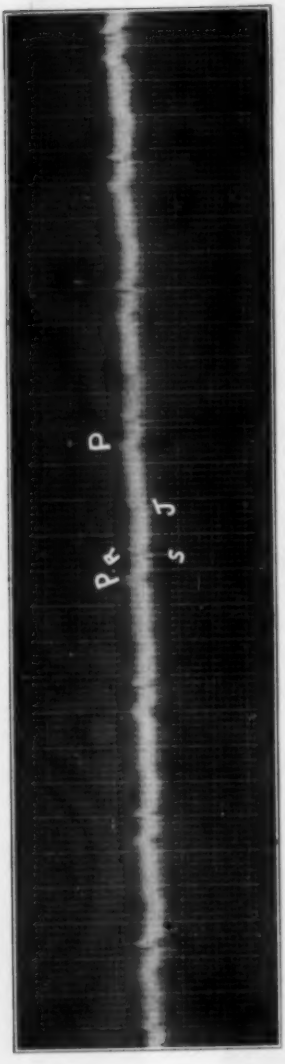

Figure 17

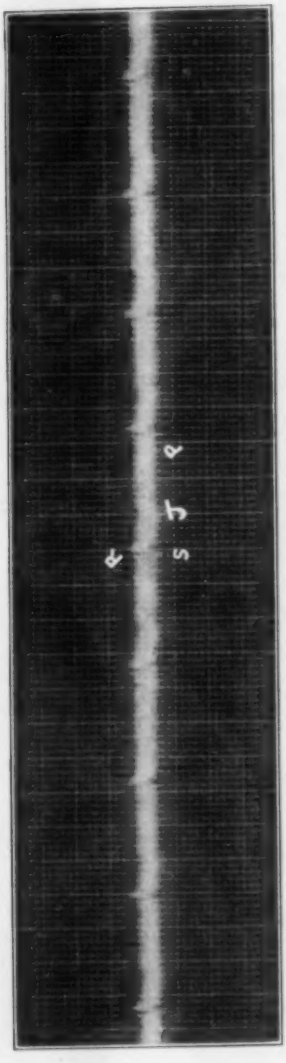

Figure 18

of auricular pathology. In this series, corroborative evidence for this view may be found in such findings as an associated rise of temperature, severe acute angina, pulsus bigeminus, etc. Migration of the pacemaker, however, *without* extrasystoles, especially when found in neurotic individuals, in the presence of prolonged nervous and mental strain, following excessive smoking, associated with disturbance of respiration, relieved (or caused?) by atropin, occurring on assuming the upright posture, is probably to be interpreted as due largely, if not entirely, to variations in vagus control.

SUMMARY

1. Eighteen cases of inversion of the P wave (migration of the pacemaker) are reported; twelve cases with normal rhythm, six cases with arrhythmia (auricular extrasystoles).

2. Inversion of P wave is most frequent in Lead III; five cases showed inversion in Leads II and III; one case in Leads I, II and III; five cases showed a diphasic P; four cases showed a bifurcated P.

3. Analysis of these cases shows that the majority of the patients suffer with varying degrees of myocardial insufficiency and have associated acute or chronic infectious processes. In addition, evidence of vagal influence is frequent, as shown by disturbances in respiration, effect of atropin, occurrence in vagotonic persons, etc.

4. Inversion of P wave *with* auricular extra systoles should probably be interpreted as evidence of auricular pathology; *without* extra systoles, is probably due to variations in vagus control.

5. Electrocardiographic study of cases showing inverted P, or of cases occurring in suspect vagotonic individuals should include the effect of (a) deep breathing, (b) change of posture, (c) atropin and (d) the effort test.

Sincere appreciation to several members of the hospital staff is herewith offered for permission to publish curves of their cases, and in particular to Miss Marguerite Cullver, laboratory technician, for invaluable aid in the preparation of this material.

AN ELECTROCARDIOGRAPHIC SIGN OF CORONARY ARTERY OBSTRUCTION

HAROLD E. B. PARDEE, M.D.
NEW YORK

The study of the form of the electrocardiogram continues to uphold the idea that when the muscular tissue of the heart is diseased, there will follow abnormal variations in the electrical currents due to the heart's contraction. It is hoped to show that obstruction of a branch of coronary artery is followed by a sign which is characteristic of this condition and is readily recognizable in the human electrocardiogram.

REPORT OF CASE

A patient was admitted to the second medical division of the New York Hospital, on the service of Dr. William R. Williams, to whom I am indebted for the privilege of publishing this case. Two hours before admission he had had an attack which was typical of the symptom complex due to an obstruction of one of the branches of a coronary artery.

He was 38 years of age. He had never had rheumatic fever or more than very occasional slight attacks of tonsillitis. Syphilitic infection was denied, though he had gonorrhea at the age of 16, followed by inguinal adenitis. Otherwise he had always been in the best of health until August, 1916, when he had a slight attack of "indigestion," with epigastric distress and eructations of gas. At this time his physician told him that he had heart trouble, although he had never had reason to suspect this himself. About December, 1916, he began to notice that his "wind" was not so good as it had been, and during February, 1917, this trouble became distinctly worse and he also noticed occasional slight aching precordial pain.

March 4, 1917, suddenly, while he was in bed, asleep, he felt a sharp stabbing pain beneath the sternum. This radiated about the left chest and down the left arm and was very severe. He felt very weak and prostrated and thought he was going to die. His heart was beating very heavily, he said, and very slowly. He was not short of breath and did not lose consciousness. After twenty or thirty minutes he vomited, and this was repeated several times during the course of the day. The pain continued severe, although less so after the vomiting.

On admission to the hospital two hours later the heart rate was 44 per minute and the rhythm was noted to be regular, though this seems doubtful in view of later findings. The apex beat was noted as of fair force, the heart sounds of poor quality. The patient appeared cyanotic and showed slightly increased respirations. He complained of precordial pain but not of dyspnea. An electrocardiogram (Fig. 1) was taken by Dr. H. J. Spencer about four hours after the initial attack. At this time his condition was practically as described on admission, and throughout the day he complained of dull precordial pain, made worse by effort, showing slightly increased respirations, but not feeling short of breath, except when lying flat in bed.

During the next two days the cyanosis, increased breathing and precordial pain gradually decreased. His temperature varied from 100 to 101 F. for three days and thereafter was normal. The heart rate varied from 60 to 80 per minute and continually showed the irregularity of auricular fibrillation. The blood pressure was estimated on several occasions and averaged about 130 mm. systolic and 80 mm. diastolic. A Wassermann test was negative.

March 7, when the next electrocardiogram (Fig 2 A) was taken, he was quite comfortable when at rest and appeared to be a practically normal man, although no exertion could be undertaken without marked palpitation and dyspnea and some precordial pain. March 10 he was allowed up in a chair, but felt so weak and was so troubled by palpitation and pain that he did not try it again. While in bed he was quite comfortable and without complaint. Figure 2 B is the record taken March 13, and Figure 2 C was taken March 20. He was up in a chair again March 16, and this time did not complain. His strength improved quite rapidly after this, so that he returned home March 23 after having been walking about the ward without symptoms of distress for the last three days.

He returned to his work as a chauffeur about April 1, and was under my observation until July 11, 1917. During this time he continued to drive his car and often drove very fast. He was also able to change tires without difficulty. It was necessary for him to take digitalis at times to ward off dyspnea on exertion which made its appearance when he stopped the drug. In June, he had another attack, which was possibly a coronary occlusion, though of a smaller artery. While lying in bed, there was a sudden sense of constriction in the chest. He became pale and had to struggle for breath. This passed off after ten or fifteen minutes, leaving him quite weak, but next morning he was all right again and able to go to work. An electrocardiogram taken five days later, was not different from those taken previous to this attack.

The clinical records subsequent to July 11, 1917 are very unsatisfactory. It appears, however, that he continued to come to the clinic at from two to four months intervals. He continued at work for most of the time though there were periods when he was prevented by dyspnea, which was noted as improved after taking digitalis for a time. His disability during the good periods did not keep him from work, and, in fact, in June, 1919, he suddenly fell dead with an attack of severe precordial pain, just after having returned from a day of driving his automobile.

DISCUSSION OF ELECTROCARDIOGRAMS

The record shown in Figure 1, taken about four hours after the beginning of the attack, has very remarkable ventricular waves. Besides the small wavelets marked f f f, due to fibrillation of the auricles, there are the large mounds in Leads II and III, which must correspond to those waves in Lead I which are marked R and T, and which bear a closer resemblance to the usual ventricular waves. It can also be seen that the large waves of Leads II and III are composed of an initial, quickly varying, sharp pointed group, followed by a slower variation, as in the normal ventricular complex. In Lead II, and more especially in Lead III, the slow variation, T, starts from a point well up on the descent of the R wave instead of, as is usually the case, from the base line after the R wave is completed. In Lead III the point of origin of the T wave is almost at the apex of R. These waves then may be divided into a Q R S group and a T wave to bring them in line with the nomenclature of the normal electrocardiogram, and to suggest that the physiologic processes which cause these waves are not essentially different from those causing the analogous parts of the normal electrocardiogram. There is evidently a Q wave in Leads

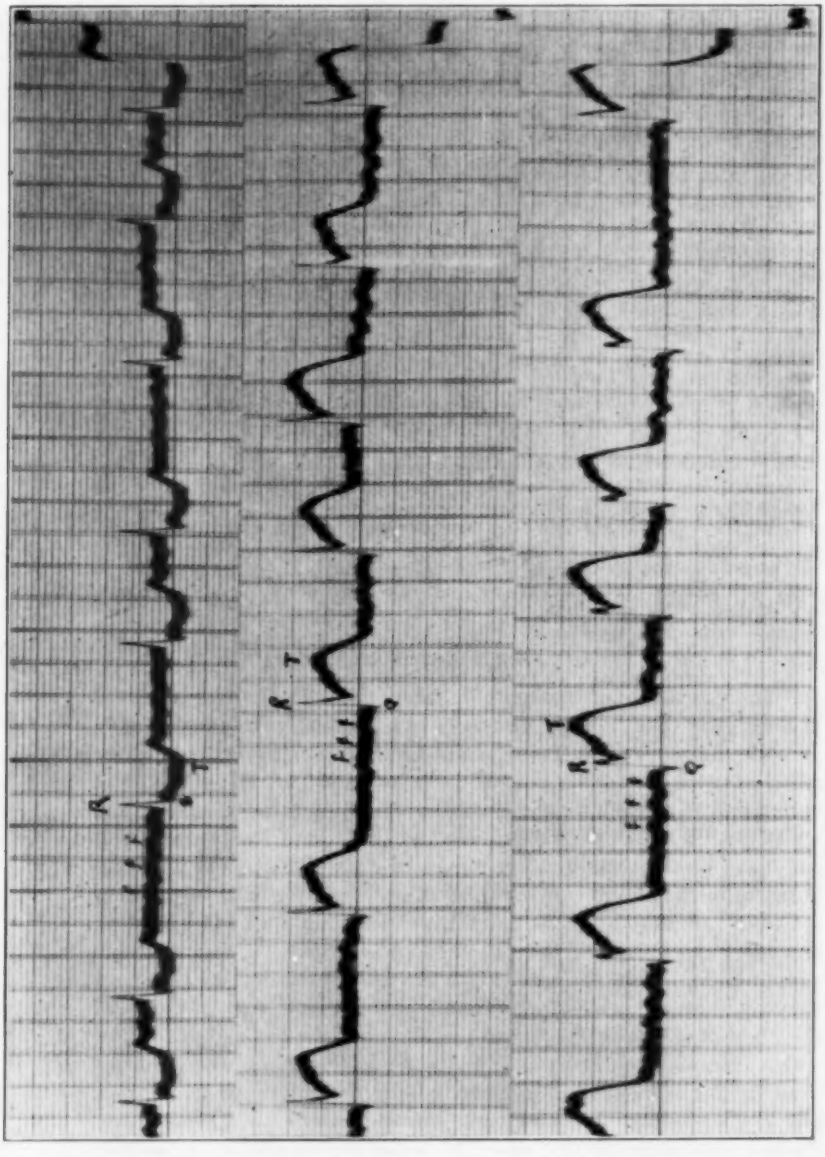

Fig. 1.—Electrocardiogram of patient, taken March 4, four hours after obstruction of coronary artery. The Leads are I, II and III from above downward. The two steps at the end of the lead are caused by introduction of 2 millivolts in the circuit with the patient, and serve as control of the standardization. The space between the corresponding line of each pair of ordinates is 0.20 second.

II and III, and an S wave in Lead I with the T starting from near its peak, as it does from near the peak of the R waves of the other leads.

The Q R S group of this record is not especially remarkable for as far as it developed, i. e., up to the point of origin of the T wave. It suggests that neither ventricle is predominantly hypertrophied. At the point of origin of the T wave there has elapsed from the beginning of this group, only 0.06 second instead of the usual 0.08 second; so it

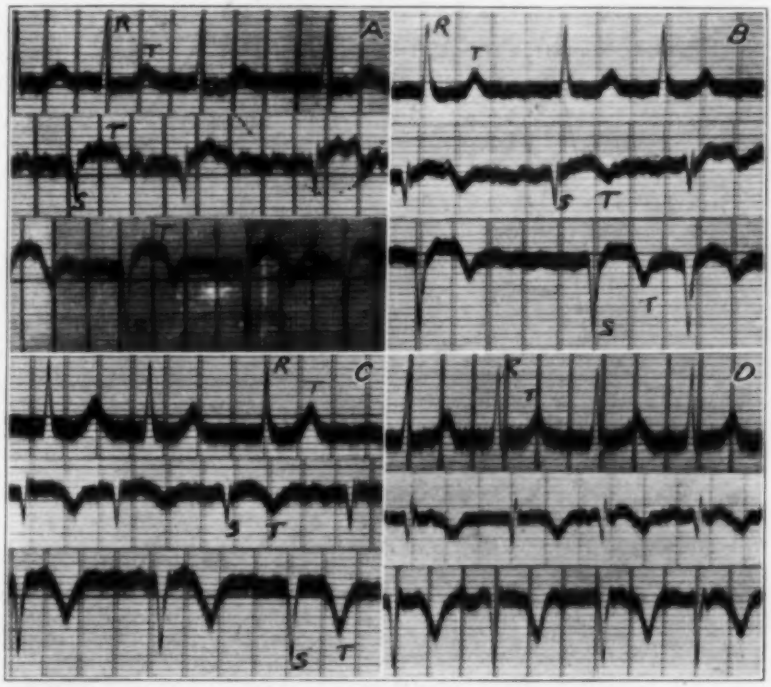

Fig. 2.—Electrocardiograms of same patient taken at later dates. A, March 7; B, March 13; C, March 20; D, May 25. The leads in each group are I, II and III from above downward.

may be said that the Q R S group was not yet completed, was, in fact, interrupted by this very unusual T wave. Whereas the T wave does not usually start until, perhaps, 0.08 second after the end of the Q R S group, and seldom reaches a height of over 5 mm., in this record the T interrupts the Q R S before its completion, lasts for almost 0.40 second and develops a height of 12 mm. in the highest lead.

EXPERIMENTAL PROOF

Such a form for the ventricular waves of an electrocardiogram is most unique and has been noticed in only two other series of curves which have been published. Eppinger and Rothberger injected silver nitrate solution into the deep layers of the left ventricular muscle of a dog and obtained by a lead from the anus and esophagus, curves which bear a very close resemblance to Lead II of Figure 1. Other curves were obtained a few minutes later very much like Lead III. These are shown in Figure 3 A. The anus-esophagus lead does not bear a very different relation to the dog's heart from the relation which Lead II bears to the human heart, so that these curves are as

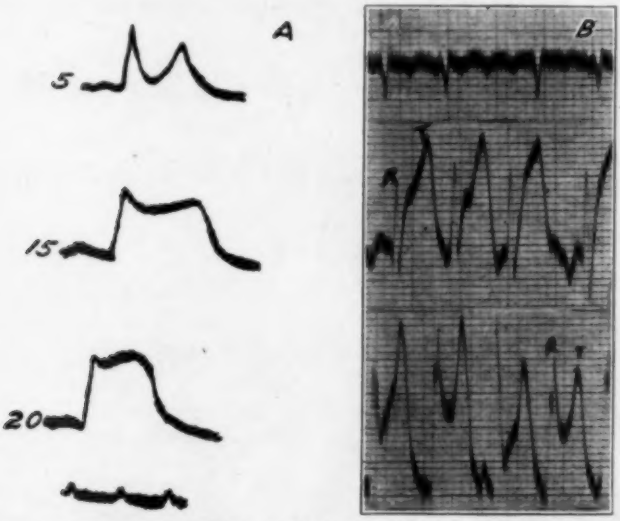

Fig. 3.—A, copy of records obtained by Eppinger and Rothberger after injection of silver nitrate into basal ventricular muscle of a dog. From above downward these curves were obtained five, fifteen and twenty minutes after the injection. 3 B, record obtained by Smith five minutes after ligation of a branch of a coronary artery of a dog. From above downward are Leads I, II and III.

comparable to ours as curves from the lower animals ever can be. They obtained curves of this sort in thirteen out of the twenty-five experiments on the left ventricle, and noticed that the first change was an increase in the height of the T wave, later records showing the origin of the T wave from an increasingly high point on the descent of the R wave, so that eventually it came off from the very peak and extended above it as in Lead III of our case.[1] For this reason it is

1. Eppinger and Rothberger: Zur Analyse des Elektrokardiogramms, Wien. klin. Wchnschr. **22**:1091 1909.

believed that this record of our case indicates coronary artery obstruction, such an obstruction of necessity being followed, as would silver nitrate injection, by an area of muscle degeneration.

More recently a curve has been published by Smith,[2] which was obtained five minutes after ligation of the ramus circumflex sinister of the heart of a dog, and which shows in Lead III the T wave coming off of the descending limb of R about half way down to the base line This curve is reproduced in Figure 3 B. Smith found that the height of the T wave was always increased immediately after the ligation of branches of the left coronary artery, and that its "height seemed to vary directly with the size of the branch ligated." The findings of Smith are in agreement with those of Eppinger and Rothberger, and strengthen the diagnosis in our case.

Injury to the musculature of the right ventricle was found by Eppinger and Rothberger [1] to cause an increase in the size of the T wave also, but the Q R S group was synchronously affected so that the result was a complex resembling that obtained by this lead after injury to the right branch of the A-V bundle in the dog, with abnormally wide Q R S group, small R, large S and large, upwardly directed, T wave. The one record after ligation of the right coronary artery which is published by Smith,[2] shows this same general type of ventricular complex.

THEORETICAL PROOF

All of these experimental facts point to the fact that our case has a large lesion in the left ventricle due to occlusion of a coronary artery, and this conclusion is upheld by two varieties of theoretical evidence from the record itself. First, the Q R S group starts as in a normal electrocardiogram with the ventricular predominance within normal limits. The departure from the normal curve comes at the peak of the S wave in Lead I, at the thickening of the descending limb of R in Lead II and in Lead III at the notching of the R wave. These curves were enlarged photographically and measured, and it was found that all of these points follow the beginning of the Q R S group by from 0.05 to 0.06 second. Lewis [3] has shown that by the time that 0.05 second has elapsed all of the muscle of the heart has entered into contraction, except that portion at the basal part of the left ventricle.

To return to our case: we note that the curve is a normal one until the time when the basal ventricular musculature becomes involved in the contraction. The second bit of evidence from the records is that

2. Smith, F. M.: The Ligation of Coronary Arteries with Electrocardiographic Study, Arch. Int. Med. **22**:8 (July) 1918.

3. Lewis: The Spread of the Excitatory Process in the Vertebrate Heart, Phil. Tr. Roy. Soc. London, Series B, **207**:221.

the T wave of this record is downward in Lead I and upward in Leads II and III, so that during this period, when all of the ventricular muscle is contracting and when in normal hearts there is practically no flow of current, we find a very large current in this patient, the predominant direction of which within the heart is toward the right as opposed to the left half of the heart, toward the apical as opposed to the basal half, and toward the lower as opposed to the upper half.[4] In Figure 4, the shaded area, projected right through the substance of the ventricles, is the only region of the heart which could produce a current fulfilling these qualifications if by reason of a much increased activity its action current should become predominant over all other currents at this time. This is thought to be the mode of production of the T wave of Figure 1, for the area affected by an arterial occlusion would become an area of anemic necrosis, and the first physiologic

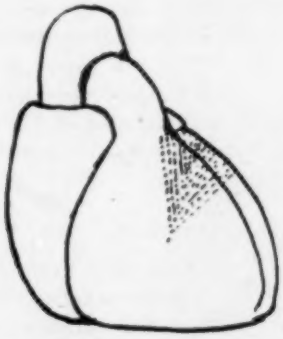

Fig. 4.—Diagram of heart showing probable region of the ventricles affected in the patient of Figures 1 and 2.

action of this would probably be a brief increase in the chemical activity of the area, although very shortly the area would cease to function. This hypothesis is supported by the findings of Eppinger and Rothberger. They found, as has been said, that silver nitrate, injected into the basal part of the left ventricle, gave a high T wave by the anus-esophagus lead, i. e., produced a predominance of current within the heart in a direction from base toward apex, but they also found that cooling of this same region by the ethyl chlorid spray gave a downwardly direct T wave,[1] i. e., a current flowing in a direction from apex to base within the heart. Cooling with ethyl chlorid would certainly lead to a diminution of the chemical processes of contraction in the area affected, and the silver nitrate had quite evidently the

4. See in this connection Einthoven, W.: Weiteres über das Elektrokardiogram, Arch. f. d. ges. Physiol. **122**:576, 1908.

opposite effect. Eppinger and Rothberger did not take records of their dogs for more than a few hours, but Smith's later records, as well as those of the case here reported, show that the T wave eventually assumes an opposite direction to that which it takes at first. The brief increase in the chemical activity is followed by a diminution.

LATER RECORDS

The next record of this case was taken March 7, three days after the attack. This is shown in Figure 2 A, and bears a closer resemblance to the usual electrocardiogram. The Q R S group shows the absence of R in Lead III, and the deep S in that lead which results from predominance of the mass of the left ventricle. This group does not show an abnormal duration, so that we may say there is no prolongation of the time taken for the spread of the contraction throughout the ventricular muscle,[5] although the notching of the upward limb of the S wave in Lead II, taken together with the broadening of this upward stroke in Lead III, indicates that there is some abnormality in the spread of the contraction wave. The T wave has changed considerably, so that in Lead I it no longer has a downward but an upward direction. In Leads II and III the former upward direction is retained but the amplitude is much smaller, and at the end of this wave at the same time interval after the Q R S group as in the peak of the upward T wave in Lead I, there appears a small downward wave in Lead III. A record taken March 13 is shown in Figure 2 B and is practically the same as that shown in Figure 2 A, except that the rate has been slowed by digitalis, and that there is a definitely downward T wave in Lead III while one is apparently developing in Lead II, as it did in Lead III, by the latter part of this deflection dipping below the line, the part immediately after the Q R S group remaining as an upward deflection. Lead III still continues to show a slight upward deflection during this period.[5]

The next record was taken March 20, and is shown in Figure 2 C. The Q R S group is unchanged from the last record, and the downward T wave is well developed in Lead II and of large size in Leads I and III, especially the latter, where it shows its maximum value. In none of the leads does the interval between Q R S and T depart far from the base line of the record, though it does so in Leads II and III. After

5. This distinction between the direction of the peak of T and that of the current before this peak, has been neglected by most writers. It should not be neglected for the current between S and T has frequently a direction different from that of the T wave itself, and of course is indicative of the muscular activity of the heart at an earlier part of the heart cycle than the peak of T. It may vary when the T wave does not, a common observation after the administration of digitalis being that this interval will be changed at a much earlier stage of the digitalis effect than the T wave itself.

this date the electrocardiogram changed very little for as long as it was observed, which was until July 11. Records taken March 19 and April 6 showed ventricular waves practically identical with those of Figure 2 C. Figure 2 D is the record taken May 25, and shows a slight change in Lead II in the Q R S group, and records taken June 29 and July 11 show waves practically identical with those of this figure.

The very prompt appearance of left ventricular predominance in this patient is difficult to explain on any other theory than the destruction of an appreciable amount of the muscle of the right ventricle. The sector of the heart indicated in Figure 4 can be seen to include left as well as right ventricle, but owing to the fact that the mass relation of the ventricles normally averages about left to right as 2.2 to 1,[6] it follows that an area of necrosis involving parts of both ventricles such as would result from obstruction of arterial branches to the septum, would cause a relative predominance of the left ventricular mass, this predominance increasing greatly for slight increases in the size of the necrotic area even though both ventricles were equally involved.

The more gradual change in the direction of the T wave may go with the more gradual establishment of complete loss of function in the necrotic area, combined with a more gradual restoration of normal function in the tissues at its border by the collateral circulation. The new direction which the T wave has taken on indicates a predominant current within the heart which flows in a direction more from the right toward the left, more from the apex toward the base and more from the lower toward the upper halves of the heart.[4] This is opposite in direction to the current which was flowing during this part of the heart cycle immediately after the injury, and probably depends on the absence of function within the injured area which was at first overactive, so that now the currents due to normal activities in other areas have become predominant. The flow is toward the injured region just as after cooling the muscle with ethyl chlorid. A similar change in the direction of the T wave of the dogs was noticed by Smith to take place twenty-four hours after the onset of the arterial obstruction, and, in fact, many of the records published by him show characteristics similar to the later records of our case shown in Figure 2.

We have, then, seen the form of the human electrocardiogram immediately after the obstruction of a branch of a coronary artery, and the form which it later assumes and maintains for a period of four months. All of the theoretical and experimental evidence which has been mentioned, is seen to be in agreement as to the site of the injured area, and thus tends to support and to prove itself.

6. Lewis, T.: Ventricular Hypertrophy, Especially Preponderance of One or Another Chamber, Heart **5**:367, 1914.

CLINICAL OBSERVATIONS

In order to find whether these observations could be made use of clinically, a large number of electrocardiograms have been gone over, looking for those with the characteristics which appeared in the later records of this patient. Four were found in about 1,000 consecutive records, and to these may be added one record published by Herrick.[7] These five electrocardiograms are shown in Figure 5. The clinical records of all of these cases, but one, included descriptions of typical attacks of anginal pain. This one patient (Case 2) complained of precordial discomfort which was felt on walking during the first hour after meals, and less frequently at other times. Figure 6 shows four other records which were selected because they showed ventricular complexes with characteristics similar to those of our case, but which were evidently not typical. None of these four patients gave histories of precordial pain.

It seems a suggestive fact that precordial pain was present in all of the cases of Figure 5 whose records closely resemble the later ones of our case shown in Figure 2, and was present in none of the cases of Figure 6 whose records do not so closely resemble those of Figure 2. In addition to this, the dogs operated on by Smith showed, after a day or two, electrocardiograms which also had characteristics similar to those of the records of Figures 2 and 5. For these reasons, I believe that when we find such an electrocardiogram from a patient with precordial pain, even of very slight degree, it is proper for us to make the diagnosis of an old obstruction of a branch of a coronary artery. The characteristic features of these records seem to be that the Q R S group is usually notched and usually shows left predominance: the T wave starts from a point near but not directly on the base line, and in one lead, either Lead III or Lead I, quickly leaves it in a sharp curve, as in Lead III of Figure 2, and Lead I of Case 6 in Figure 5, there being no short level stretch before the peak of T as is so evident in normal records; the T wave is usually turned downward in two of the three leads, in Lead II and either Lead I or Lead III; the T wave is usually of large size and accordingly has a sharper peak than usual. Records taken shortly after the obstruction has occurred will not have these characteristics, but will be like that of Figure 1, the typical features being the extreme height of the T wave, and the fact that this wave starts from a point on the Q R S group well away from the base line, denoting that the normal balance of the currents in the heart which should occur at this time, has not taken place.

7. Herrick, J. B.: Thrombosis of the Coronary Arteries, J. A. M. A. **72**:387 (Feb. 8) 1919.

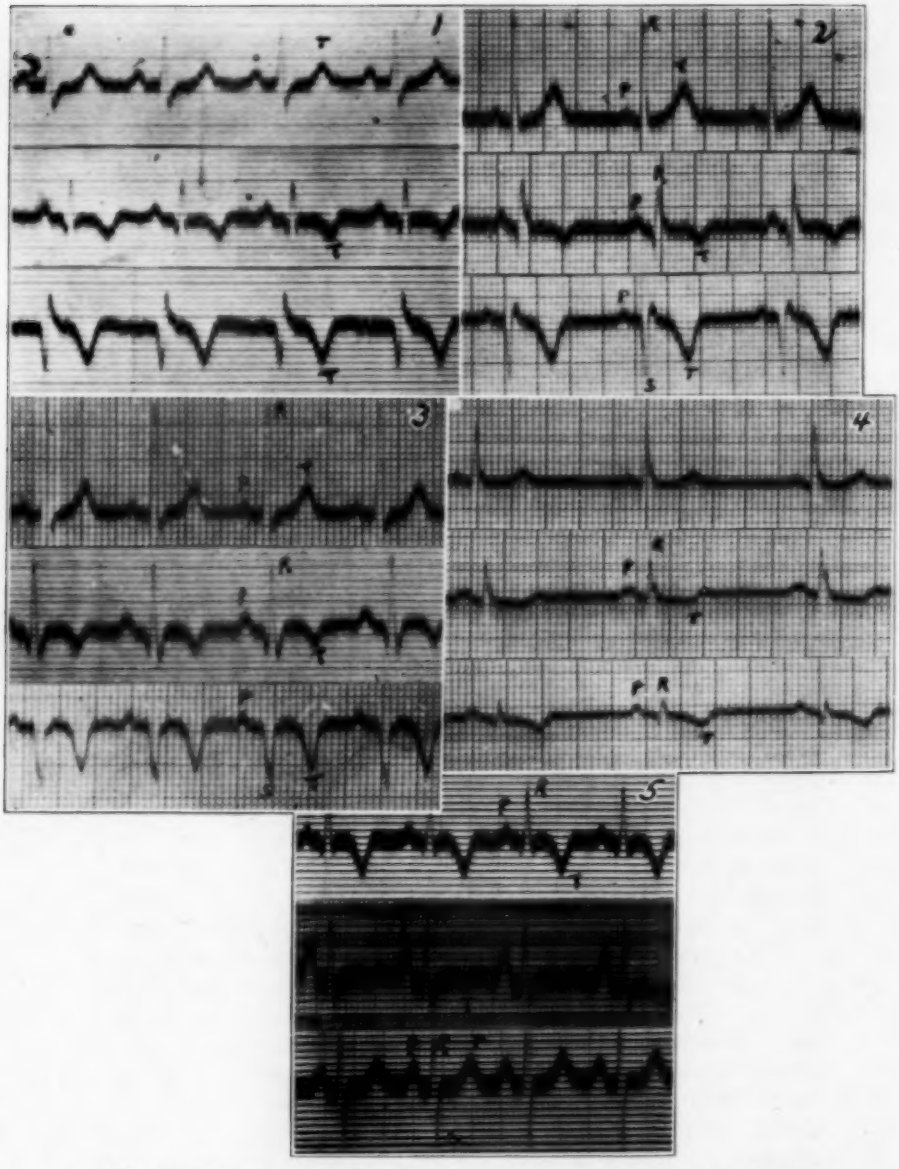

Fig. 5.—Electrocardiogram of five patients, all of which show the changes resulting from former coronary artery occlusion. Record No. 5 is of Herrick's case. (Through the kindness of Dr. Fred M. Smith, who was good enough to send me the prints, I am able to publish this curve and also that of Figure 3 B.)

It is reasonable to suppose that a coronary obstruction may arise gradually by a narrowing of the lumen of the vessel by arteriosclerosis, and lead to the same sort of an area of degenerated muscle as is found some time after the occurrence of an acute obstruction, and that such a case would give the typical electrocardiographic signs. This may account for the fact that Case 2, in Figure 5, shows these signs without having had a severe acute attack such as would accompany an acute obstruction. Further, it is likely that any other disease condition, such as syphilis, which leads to the degeneration of a large localized area of ventricular muscle, would also give an electrocardiographic picture of this sort, for there is nothing special about coronary obstruction in relation to this sign, except the relatively great frequency of its occurrence as a cause for localized areas of myocardial degeneration.

It is true, that not all patients who have attacks of precordial pain show this sign, but this does not prove its unreliability, for there are other causes for attacks of precordial pain than coronary obstruction. It seems likely, also, that a certain minimal area must be involved in order to affect the electrocordiagram in this characteristic way, and that involvement of a lesser area might still cause pain. The only case in which a necropsy was made is that of Herrick, and this heart showed relatively large areas of myosclerosis, one being "4 cm. in diameter" on the outer wall of the left ventricle, and another involving "the wall of the left ventricle near the apex, and the interventricular septum." The apical involvement of the ventricles in this case is of interest in connection with the fact that the T wave of his electrocardiogram (No. 5 of Fig. 5) is caused by a current with a direction opposite to that of the current of the T wave of Figure 2 which has been conconsidered to have the involvement in the basal region of the ventricles.

These signs which have been described may very well not be the only ones which are produced by coronary disease, but I believe that the evidence that they are so produced is reasonably good. They seem to indicate obstruction in the area supplied by the left coronary artery, though this is not conclusively proven. It seems likely, also, to judge from the experimental work which has been mentioned, that some of the electrocardiograms which have the form characteristic of a lesion of the right branch of the auriculoventricular bundle, are, in fact, due to obstruction in the area supplied by the right branch of the coronary artery. Both of the references cited show such records, obtained after lesions affecting this area, and theoretical considerations would lead us to expect this form after such a lesion.

SUMMARY

1. A patient who had just had an attack typical of occlusion of a coronary artery showed a very remarkable electrocardiogram.

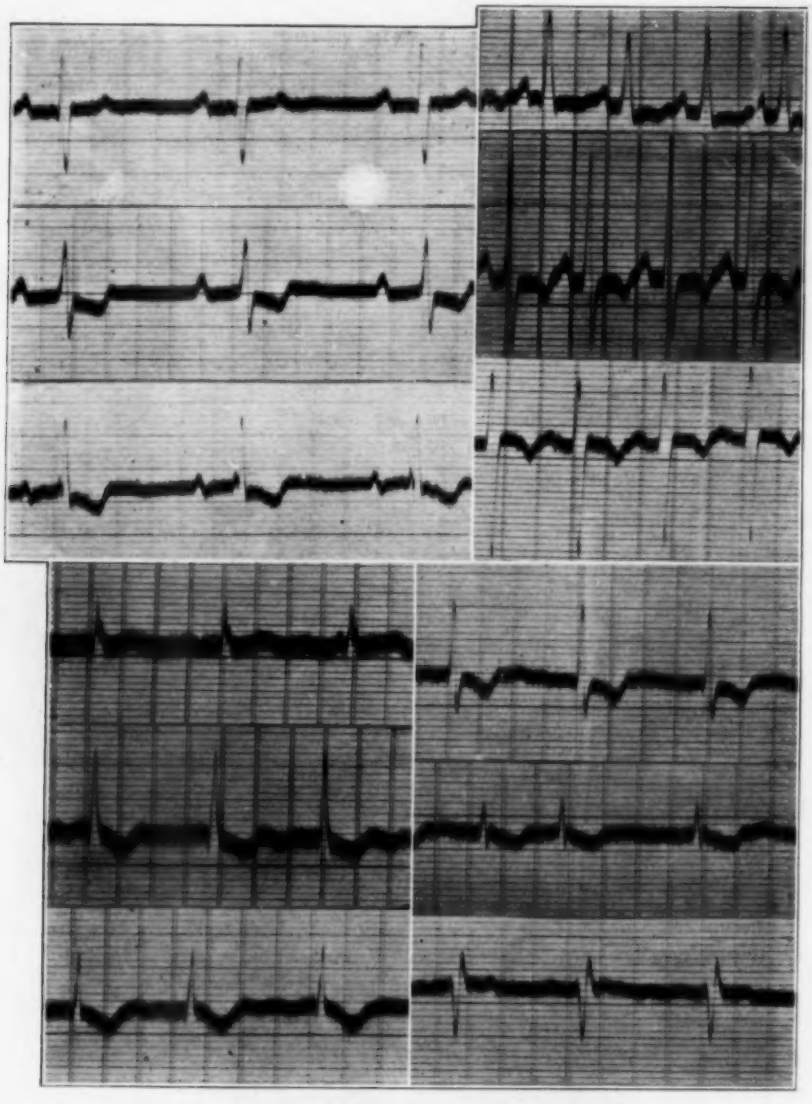

Fig. 6.—Electrocardiograms of four patients, all of which show changes in the ventricular waves but not those typical of coronary occlusion.

2. The patient recovered and the electrocardiogram had changed on the fourth day to a form which it retained for the four months during which he remained under observation. He lived for two years longer, and died with a typical attack of angina pectoris.

3. Five other records have been found which showed characteristics like those of the later records of this patient. Four of these five patients gave a history of typical attacks of anginal pain, while the other had slight precordial pain on exertion. One case, which came to necropsy, showed large areas of muscle degeneration at the apex of the ventricles.

4. The form of the electrocardiogram in these cases is what might be expected on theoretical grounds to result from the occurrence of a fairly large area of degeneration in the ventricular muscle.

5. Experimental work on dogs agrees with the theoretical considerations, and has produced changes in the dogs' records which are similar to those in the case here reported both at the time of the obstruction and later.

6. The characteristic changes appearing a day or two after the obstruction are as follows: The Q R S group is usually notched in at least two leads, and usually shows left ventricular predominance. The T wave does not start from the zero level of the record in either Lead I or Lead III though, perhaps, from a level not far removed from it, and in this lead quickly turns away from its starting point in a sharp curve, without the short straight stretch which is so evident in normal records preceding the peak of the T wave. The T wave is usually of larger size than customary and accordingly shows a somewhat sharper peak. The T wave is usually turned downward in Lead II and in one other lead. Not all of these changes are to be found in every record, but enough of them are present to give it a characteristic appearance.

7. It is concluded that this electrocardiographic sign indicates the presence of a rather large area of muscle degeneration, and when it is obtained from a patient who gives a history of precordial pain coming either in attacks or upon exertion, that it will complete the diagnosis of obstruction of a branch of a coronary artery.

8. It is suggested that this sign results only from a lesion within the area supplied by the left coronary artery, and that one within the area of the right coronary artery would cause changes so that the record would more or less closely resemble that which follows a lesion of the right branch of the auriculoventricular bundle.

BOOK REVIEWS

A LABORATORY SYLLABUS OF CLINICAL PATHOLOGY. By CHARLES E. SIMON, B.A., M.D., Professor of Clinical Pathology in the School of Medicine and the College of Physicians and Surgeons of the University of Maryland, Baltimore. Ed. 1. Cloth. Pp. 86. Philadelphia: Lea & Febiger, 1919.

The author presents this manual for several reasons, which, during his large experience in teaching clinical pathology, have become more and more evident to him. One of the chief reasons for this manual is to give a method whereby the brief half-hour talks by the instructor at the beginning of each period can be eliminated, thereby saving wasted effort on the part of the instructor and providing the student with more time to study and complete the work of each period. The laboratory work has been arranged on the basis of thirty-nine lessons, each lesson to occupy a two-hour period. The subject matter has been arranged in connection with each lesson under three headings, viz.; (1) Instructions to laboratory assistants, regarding material, reagents, etc. (2) Instructions to the student as to method and manner of reports, etc. (3) A set of questions based on the work done in the laboratory and on home reading, which are to be answered in writing at home and presented as part of the work done in the course.

The scope of the work covered is quite extensive, the methods well selected and in general use, and it places the burden of supplying a large variety of material on the instructor, which is as it should be. Most physicians, as well as students, would profit greatly if they could have the opportunity of taking such a course. The book should be found serviceable as a teaching manual, but would be of limited value to the practicing physician.

COLLECTED STUDIES ON THE PATHOLOGY OF WAR GAS POISONING. From the Department of Pathology and Bacteriology, Medical Service Section, Chemical Welfare Service, Under the Direction of MAJOR W. C. WINTERNITZ. New Haven: Yale University Press, 1920.

The present volume is the first work published by the Yale University Press on the Anthony N. Brady Memorial Foundation. Three distinct lines of work were undertaken by the Medical Service Section—physiology and toxicology, metabolism and pathology. This thesis represents the work of the last named group.

The studies are presented in a large volume with a profusion of illustrations, many of these done in colors. Indeed one might obtain a very good idea of the effect of poison gases on the air passages and lungs simply by a careful inspection of these remarkable drawings and water colors.

One chapter is devoted to an interesting experimental study of intratracheal therapy. The authors found that perfusion of the lung with large volumes of salt solution is effective in removing such material as India ink, starch paste and bacteria. The experiments suggest the possibility of intratracheal therapy in gas poisoning based on pulmonary irrigation.

Attention is called to the tendency to hemorrhage in the lungs for weeks and months after gassing. The animals that died a violent death showed a higher percentage of hemorrhage than those that died of disease. The authors conclude that a soldier recovered from the acute effects of gassing should be regarded as an individual with damaged lungs and should avoid muscular effort or other practices which might lead to increased blood pressure. Otherwise pulmonary hemorrhages are likely to occur.

This monograph is altogether a most creditable and important contribution to war medicine.